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THE UNIVERSITY OF ALBERTA

THE EFFECT OF INTENSITY OF EXERCISE ON BODY COMPOSITION CHANGES IN WOMEN

by

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A THESIS
SUBMITTED TO THE FACULTY OF GRADUATE STUDIES
AND RESEARCH IN PARTIAL FULFILMENT OF THE
REQUIREMENTS FOR THE DEGREE OF MASTER OF SCIENCE

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THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled THE EFFECT OF INTENSITY OF EXERCISE ON BODY COMPOSITION CHANGES IN WOMEN submitted by R. ANN MALSBURY in partial fulfillment of the requirements for the degree of Master of Science in Physical Education.

Patricia R. Conger
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Date January 23, 1980

Dedicated in memory of my grandparents,

William John and Isabella de Grotte Malsbury

ABSTRACT

The purpose of this study was to investigate the effects of high and low relative intensities of exercise on body composition changes of women. Another purpose was to examine the effect of training at high and low relative intensities on resting and post exercise glucose, lactate and FFA concentrations.

Thirty-four female subjects were ranked according to percent body fat and $\dot{V}O_2$ max and were randomly assigned to either a high intensity group which worked at 80% of maximum heart rate, a low intensity group which worked at 65% of maximum heart rate or a control group who maintained their normal exercise and eating habits. The training program consisted of three training sessions per week for nine weeks. Groups were equated on total power output of 12,000 kpm for four weeks after which it was increased to 14,000 kpm.

Both the high and low intensity groups showed a significant decrease in percent body fat ($p < 0.05$) and an increase in fat free weight ($p < 0.05$) while the control group showed no significant changes. There was no difference ($p > 0.05$) between the high and low intensity exercise groups in changes in percent body fat and fat free weight. The low intensity exercise group lost 2.9 lb of fat ($p < 0.05$) while the high intensity group lost 2.4 lb of fat ($p > 0.05$). There was no decrease ($p > 0.05$) in total body weight for any of the groups. $\dot{V}O_2$ max did not change significantly ($p > 0.05$) in any of the groups.

Resting lactate levels remained the same for all groups from pre to post training. Post training lactate values of the trained subjects remained the same as pre training levels but were produced at a higher total power output. The control groups produced more lactate than the exercise groups post training ($p < 0.05$).

Both the high and low intensity exercise groups showed similar decreases in resting and post exercise levels of FFA after training ($p < 0.05$).

Resting glucose values were lower after training for both the high and low intensity exercise and control groups ($p < 0.05$). The high and low intensity control groups had decreased post exercise glucose values from pre to post training tests ($p < 0.05$).

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CHAPTER I

INTRODUCTION

Repeated aerobic exercise is associated with adaptations in body composition which are reflected in decreased body fat and a concomitant increase in fat free weight (Parizkova, 1963; Moody et al., 1969; Moody et al., 1972; Smith, 1975; Bjorntorp, 1975; Zuti, 1976; Smith, 1976; Novak, 1978). Changes in body composition are dependent on the separate effects of repeated exercise sessions on muscle, adipose tissue and metabolism. It may well be that metabolic and endocrine adaptations occur first and are later reflected in body composition changes. In order to prescribe exercise as an effective part of a weight reduction program, the physiological adaptations caused by specific exercise programs must be considered.

Physical training produces a decrease in body fat which implies that training altered adipose tissue metabolism towards increased lipid mobilization and/or decreased lipid assimilation (Kral, 1974; Holm, 1977). Exercise increases the need for fuel to supply energy for muscular contractions. During aerobic exercise, the increase in fuel supply is met from a combination of carbohydrates and fat. The mixture of fuels used will depend upon the fitness level and prenutrition of the subject as well as the intensity and duration of exercise (Christensen and Hansen, 1939; Issekutz, 1963; Havel, 1971; Astrand, 1977; McCafferty and Horvath, 1977; Wahren, 1977; Vranic and Berger, 1979). During moderate intensity exercise in which there is little increase in blood lactate levels, the utilization of FFA as a fuel may provide 25-90% of the energy produced (Paul and Holmes, 1975; Sutton, 1978). Exercise causes an increase in FFA turnover rate, the amount being dependent on the intensity of exercise relative to the individual's fitness level (Pruett, 1970). As exercise becomes more severe in relation to the individual's $\dot{V}O_2$ max, a larger proportion of energy is derived anaerobically and the mobilization of FFA is depressed by high lactate concentrations (Pruett, 1970; Boyd et al., 1974). Thus, it would appear that intensity of exercise is a key factor affecting FFA mobilization.

The Problem

Adjustments in metabolism and body composition which result from exercise form the feasibility of physical training as a means of therapy in obesity. From a practical point of view, it is desirable to know what quantity of exercise is needed to produce body composition changes. Several researchers have indicated that intensity of exercise is a key factor in producing metabolic changes which in turn affect adipose tissue depots (Parizkova, 1963; Bjorntorp, 1976; Girandola, 1976; Pollock, 1977). Many studies have examined the effect of aerobic exercise on the body composition of women but few attempts have been made to quantify the intensity or total power output of work required to produce changes in body composition. Smith (1975) found no difference in body composition changes of women produced by low and high intensity endurance training. Girandola (1976) found low intensity exercise to result in body composition changes while high intensity exercise produced no changes. The effect of intensity of exercise on body composition is not clearly defined. Therefore, the problem of this study was to determine the effects of training at high or low relative intensities on body composition changes of women. Another problem was to examine the effect of training at high and low relative intensities on glucose, lactate and FFA concentrations at rest and immediately after exercise.

Definition of Terms

Maximal oxygen uptake ($\dot{V}O_2$ max): a term referring to the maximal volume of oxygen which can be consumed per minute (litres/minute or ml/kg/min)

Maximal heart rate: the highest heart rate recorded during a maximal stress test

Power output: refers to performance of work expressed per unit of time (e.g., kpm/min)

Training program: refers to a nine week period of exercising three times per week

Training session: refers to one exercise period

High intensity exercise group: refers to a group of subjects who trained at 80% of maximal heart rate

Low intensity exercise group: refers to a group of subjects who trained at 65% of maximal heart rate

High intensity control group: subjects did not participate in the training program but performed a high intensity exercise session at the beginning and end of the training program

Low intensity control group; subjects did not participate in the training program but performed a low intensity exercise session at the beginning and end of the training program

Fat free weight: sum of all the tissues of the body minus the total body fat

Percent body fat: is the percent of total body weight expressed as fat

Body composition: the combination of bone tissue, musculature and fat layers of the human body

Sedentary: a lifestyle which involves very little regular physical activity

Pre exercise: in a resting state before the start of exercise

Post exercise: during the recovery period after a training session

Pre training: refers to the time period before the start of the training program

Post training: refers to the time period immediately after the training program

Respiratory Exchange Ratio (RER): the ratio of the volume of carbon dioxide expired per minute ($\dot{V}CO_2$) to the volume of oxygen consumed during the same time interval ($\dot{V}O_2$)

ANOV: analysis of variance

Kilopond meter (kpm): is a unit of work. A kilopond is the force required to maintain a 1 kg mass against gravity and is equal to 9.8 Newtons

Watt (W): a unit of power equal to Joules/sec

Limitations

1. Residual volume was predicted from vital capacity.
2. Diet was not controlled. It was left to the cooperation of the subjects to maintain their normal diet and exercise patterns.

3. The control group served as a double control group as they performed both a high and low intensity work session pre and post training to obtain resting and post exercise blood samples.

Delimitations

1. Thirty-four female subjects from the university community who ranged in age from twenty-one to thirty-four years ($\bar{X} = 24.90 \pm 3.73$) participated in a nine week study.
2. Two intensities of exercise were studied.

Hypotheses

The following null hypotheses were tested for significance at the 0.05 level of probability:

1. There is no difference in the effect of high and low relative intensities of exercise equated on total power output on the body composition changes of women.
2. There is no difference in the effect of high and low relative intensities of exercise equated on total power output on resting and post exercise levels of glucose, lactate and FFA.

CHAPTER II

REVIEW OF LITERATURE

There are many aspects to the effect of training on body composition and metabolism which will be presented under the following headings: the effect of exercise on body composition, the interrelationship of FFA and glucose as a fuel, FFA, glucose, lactate.

The Effect of Exercise on Body Composition

Obesity is the end result of an energy imbalance between caloric intake and energy expenditure. Intake is the function of food consumption while energy expenditure is the sum of basal metabolic rate, specific dynamic action and the energy involved in voluntary muscular work (Garrow, 1974). Basal energy needs account for a large percentage of total caloric requirements in sedentary individuals. Although energy imbalance results when caloric intake exceeds physiological need, research indicates that the energy imbalance leading to obesity is due to lowered activity levels rather than increased food intake. Studies on obese adolescent males (Stefanik, 1959), obese adolescent females (Johnson et al., 1956), and obese adult men and women (Bloom, 1967; Curtis, 1971; Keys, 1970; Mayer, 1968) showed that obese subjects had lower activity levels but equivalent or lower caloric intakes when compared to non-obese controls. Physical activity is an important factor in producing energy balance.

By comparing trained to untrained individuals of the same average weight and height, it was found that the trained individuals had a higher body density and a lower percentage of body fat than the untrained individuals (Parizkova, 1963; Bjorntorp, 1975; Novak, 1978). It was concluded from these studies that lean body mass and fat depots are in a dynamic state of equilibrium which reflects changes in energy output and balance. During increased muscular activity, lean body mass hypertrophies while body fat decreases.

Duddleston (1970) investigated the effect of diet and exercise on obese women. The six week exercise program consisted of exercising one hour on a bicycle ergometer or treadmill four times per week. Twelve female subjects were divided into four groups. The exercise and diet group lost 15.3 pounds, the diet group lost 14.5 pounds and the exercise group lost 3.5 pounds. It was concluded that the combination of exercise and diet was most beneficial in promoting weight loss.

Johnson (1972) studied the effect of a ten week exercise program on dietary intake and body composition. Subjects pedalled a bicycle ergometer five times a week for thirty minutes with the intensity of work being determined by each individual. There was no significant decrease in body weight ($p > 0.05$); however, there were significant decreases in all skinfold measurements ($p < 0.05$), a significant increase in estimated body density and a decrease in estimated body fat of 2.2% ($p < 0.05$). Mean daily caloric intake decreased 167 Kcal ($p < 0.05$).

Kenrick (1972) evaluated the addition of exercise to a weight reduction program of twelve matched obese subjects who were on a 1000-1500 calorie diet. Six participated in a thirty minute exercise program for six months. The program included exercising on a treadmill at a heart rate of 120 to 140 beats per minute. The exercise group had a significantly greater fat weight loss than the nonexercised group.

Lewis (1976) investigated the effect of a seventeen week physical activity program on weight reduction in obese middle-aged women. Body composition was determined by hydrostatic weighing. The women participated twice a week in an exercise program which consisted of several minutes of flexibility exercises followed by nineteen minutes of walk-jogging. The intensity of exercise was maintained at 80% of the age adjusted maximum heart rate. An additional two days per week involved one hour sessions in body mechanics classes during which calisthenics, stretching and flexibility exercises were performed. Diet was modified. Significant decreases were obtained in total body weight ($p < 0.001$), fat body weight ($p < 0.001$), percent body fat ($p < 0.001$) while body density increased as did lean body mass ($p > 0.05$). Approximately 60% of the total energy deficit was attributed to a reduced caloric intake.

Zuti (1976) compared the effect of exercise, diet or a combination of exercise and diet on weight reduction of young women. Body composition changes were determined by hydrostatic weighing. The diet group reduced caloric intake by 500 calories per day; the exercise group increased caloric expenditure by 500 calories per day; the combination group reduced caloric intake by 250 calories per day and increased energy expenditure by 250 calories per day. The exercise program involved an hour of exercise equal to 250 calories (type not specified) five days per week. The exercise group was tested to determine the energy cost of walking and were prescribed a walking program equal to 250 calories. The average weight losses for the three groups were 11.7, 12.0 and 10.6 pounds, respectively, with there being no significant difference between groups ($p > 0.05$). The three groups showed an increase in body density (< 0.01). The women in the exercise and combination groups had a greater increase in body density than the diet group ($p < 0.05$) but there was no difference between the two groups ($p > 0.05$). The diet group lost lean tissue, whereas the women in the other groups showed a slight gain ($p > 0.05$). The exercise and combination groups lost more pounds of fat than the diet group ($p < 0.05$). It was concluded that if a person exercises during weight loss, fat loss is increased as well as lean body mass.

Exercise plays an important role in weight control programs by promoting optimal changes in body composition which result in a decrease in body fat and an increase in lean body mass. Some studies have researched the effects of specific exercise programs on body composition.

The effect of intense exercise on the body composition of competitive gymnasts was studied by Parizkova (1963). After sixteen weeks of intense training, body weight remained the same but the amount of subcutaneous fat decreased while lean body mass increased. Skinfold measurements were used to predict body density. Following a fifteen week period of relative inactivity, body weight increased reflecting an accumulation of fat. It was concluded that intense training caused an increase in the proportion of lean body mass with a concomitant reduction in total body fat and the opposite effect occurred with the cessation of training. Changes in body composition are more marked in athletes involved in endurance sports than in normal individuals pursuing activities involving average amounts of muscular activity.

Moody (1969) researched the effect of an eight week exercise program on body weight and skinfold thickness in overweight college women on an ab libitum diet. Energy expenditure of walking and jogging was determined and then an individualized program which required 500 Kcal/day was prescribed. Body weight decreased 2.4 kg ($p < 0.01$); percent body fat decreased 10.1% ($p < 0.01$) while lean body mass increased 2.9 kg ($p < 0.01$). Because the subjects lost more weight than would be expected from the calculated energy expenditure, it was concluded that skinfolds alone are not a valid means of measuring body composition changes.

Moody (1972) studied the effect of a jogging program on obese and nonobese teenage girls. The exercise program initially consisted of jogging and walking one mile in equal portions and was increased to three miles of 75% jogging. The obese group showed an increase in density and lean body weight. Percent body fat and body weight decreased ($p < 0.05$) in the obese girls. The nonobese girls followed the same trend although the changes were not significant.

Gwinup (1975) studied the effect of exercise on obese women over a one year period. Subjects selected an activity of their choice and increased their activity time to at least one half hour per day. The average weight loss for the group after a one year period was twenty-two pounds. It was concluded that exercise alone can produce substantial and sustained weight loss in individuals who exercise on a regular basis.

Wallace (1975) studied the effect of cardiovascular training on the body composition of college women. The sixteen week training program consisted of running and walking ten to twenty minutes three times per week at 80% of maximum heart rate. Body composition was estimated by skinfold measurements. The group lost a small amount of weight ($p > 0.05$) and a small percentage of body fat ($p > 0.05$).

A short term jogging program at two different submaximal intensities produced a decrease in percent body fat ($p < 0.05$) for both intensities as measured by skinfold measurements but showed no difference between the groups ($p > 0.05$) (Smith, 1975). Neither total body weight or lean body weight changed significantly ($p > 0.05$). The women jogged a distance of 1.75 miles at 80-85% or 70-75% of their predicted maximum heart rate for nine weeks.

Girandola (1976) researched the effect of high and low intensity exercise on the body composition of women. Training consisted of riding a bicycle ergometer three times per week for ten weeks. The high intensity group alternately exercised and rested for one minute intervals working at 840 kpm per minute for a period of five minutes. The low intensity group exercised continuously at 420 kpm/min for ten minutes. Training times were increased to seven and one half minutes and fifteen minutes by the seventh week of training. Body composition was estimated by hydrostatic weighing. After training, the high intensity group showed a slight increase in percent body fat, total body weight and lean body mass ($p > 0.05$). The low intensity group lost 1.1% body fat ($p < 0.05$), had a slight increase in lean body mass ($p > 0.05$), and a small decrease in total body weight ($p > 0.05$). It was concluded that low intensity work was the most effective type of exercise to promote body composition changes.

Smith (1976) examined the effect of seven weeks of training and seven weeks of detraining on the body composition of young women. Body composition was analysed by skinfolds. The exercise program consisted of riding a bicycle ergometer for sixteen minutes at 75% of maximum heart rate three times per week. Body weight and lean body weight increased following training ($p < 0.01$). Body fat decreased 0.3% ($p > 0.01$). Lean body weight decreased following detraining ($p < 0.01$).

Davies (1977) studied the effect of three fifteen minute sessions per week of calisthenics and running on women. Body composition was measured using skinfolds. Intensity of exercise was initially 60% of age predicted maximum heart rate and after three weeks was increased to 80% maximum heart rate. Body weight did not change ($p > 0.05$) although the sum of skinfold measurements decreased ($p < 0.05$).

Shire (1977) compared the effects of high and low resistance bicycle ergometer training on body composition. The mean total mechanical resistance was the same for the high and low resistance exercise groups. No significant changes were found in body composition as predicted from skinfold measurements.

Following a twelve week fitness class of calesthenics and endurance activities, young women lost 0.72% fat ($p > 0.05$) but showed no decrease in total body weight (White et al., 1978). Body density was estimated from skinfold measurements.

Summary

Submaximal exercise performed on a regular basis varying from three to six times per week has been shown to produce changes in body composition resulting in an increase in fat free weight with a concomitant decrease in percent body fat. Short term periods of exercise (six to ten weeks) usually result in no change in body weight due to changes in body composition. A decrease in body weight may occur with training periods of longer duration such as six months to one year. The capacity of muscle mass to hypertrophy is limited and at some time is overcome by a fat decrease which result in a net body weight decrease.

Decreases in body fat seem to occur without any voluntary restriction of energy intake; however, training effects on body composition are more pronounced if combined with caloric restriction. Exercise or a combination of diet and exercise promote a more optimal change in body composition than diet alone.

The changes in body composition which occur from exercise are dependent upon the intensity, duration and frequency of training. The type of exercise used in most training programs is aerobic work. It has not been established which combination of intensity, duration and frequency of training are required to produce optimal changes in body composition, although intensity may be a key factor.

The Interrelationship of Glucose and FFA as Fuel

During aerobic exercise, a combination of both carbohydrates and free fatty acids (FFA) are oxidized to meet energy needs (Felig and Wahren, 1975; Astrand, 1977). Early research suggested that carbohydrates were the only fuel oxidzed by the muscle (Chauveau, 1896). Measurement of the respiratory exchange rate (RER) in dogs and man implied that both fats and carbohydrates served as muscular fuel (Anderson and Luck, 1917; Cathcart and Burnett, 1925; Krogh and Lindhard, 1920). Lehninger (1946) established the oxidation of free fatty acids in skeletal muscle by determining the uptake and degradation of ^{14}C-labelled fatty acids. The relative contribution of carbohydrates and FFA as a fuel depends upon the intensity of exercise, the duration of work performed, the degree of training and pre-nutrition of the subject (Christensen and Hansen, 1939; Issekutz, 1966; Havel, 1971; Astrand, 1977; McCafferty and Horvath, 1977; Wahren, 1977; Vranic and Berger, 1979).

FFA

Fat is available to tissue in several forms - free fatty acids (FFA), triglycerides, and ketone bodies with FFA being the major supply (Paul and Holmes, 1975; McCafferty and Horvath, 1977; Newsholme, 1978). FFA originate primarily from triglycerides stored in adipose tissue (Gordon and Cherkes, 1956; Dole, 1956) although both intramuscular and extramuscular fat depots may provide FFA for oxidation (Paul and Holmes, 1975; Essen, 1977). Triglycerides are hydrolyzed to produce FFA and glycerol which are released from adipose tissue in response to hormonal and neural stimuli (Hagenfeldt, 1979).

Human FFA fraction carried in the blood is composed of a variety of saturated and unsaturated acids with chain lengths varying from 12 to 22 carbon atoms (Vihko et al., 1973). Palmitic and oleic acid make up approximately 60% of total FFA (Hagenfeldt, 1979). FFA are insoluble in water and are carried in the blood as a lipid albumin complex. Because FFA depends on albumin for transport, an upper limit of 3.5 mM/l of FFA can be carried at any one time (Gollnick, 1978).

Fasting resting levels of FFA range from 0.13 to 1.21 mM/l (Dole, 1956; Gordon and Cherkes, 1956; Foster, 1978). The use of oral contraceptives causes a decrease in resting FFA levels (Briggs, 1976). Both resting and exercise levels of FFA are affected by diet. Plasma FFA are higher after a high fat, low carbohydrate diet and, conversely, lower after a high carbohydrate diet (Astrand, 1977; Maughan et al., 1978). On a low carbohydrate diet plasma FFA concentrations increased significantly more than on a normal or high carbohydrate diet during both exercise and recovery (Maughan et al., 1978).

The FFA pool in the blood is small and FFA have a half life of 1.8 to 3.9 minutes (Eaton et al., 1969). In order to meet the needs of the cells, FFA have a rapid turnover rate (Armstrong, 1961). FFA turnover rate consists of:

1. release of FFA from adipose tissue
2. plasma concentration of FFA
3. uptake by cells
4. utilization (Rottini et al., 1971; Newsholme, 1978).

Changes in the concentration of FFA indicate only an imbalance between rate of release of FFA from adipose tissue and rate of removal by working muscle (Issekutz et al., 1966). Measurement of plasma FFA turnover rate was determined by the infusion of ^{14}C-labelled fatty acids. Armstrong (1961) established that turnover rate of FFA was related to its plasma concentration and entered cells by a mass action affect, i.e., the greater the concentration of FFA the greater the turnover rate.

It was found that plasma FFA levels were elevated during prolonged endurance exercise (Friedberg, 1963; Isskutz, 1966; Paul and Holmes, 1975). Measurement of FFA turnover rate and oxidation of isotopic FFA led to estimates that FFA could account for 25% to 90% of the total exercise metabolism (Havel, 1971; Issekutz, 1963).

FFA are oxidized to acetyl CoA in the mitochondria of the cell by the process of beta oxidation (Stryer, 1975). The rate of oxidation is dependent on the concentration of FFA to which the muscles are exposed, the ability to transport FFA into the mitochondria and the capacity of the cell to oxidize FFA. Uptake of FFA by the muscle is a function of arterial FFA concentration. When the metabolic rate is constant (at rest or during steady state exercise), the rate of fat oxidation increases with increasing FFA concentration (Armstrong, 1961; Holloszy, 1977). It appears that the availability of FFA to the mitochondria is the rate limiting factor for FFA oxidation at any given oxygen uptake (Wenger and Reed, 1976). Once in the cell, FFA must be esterified with carnitine for transport into the mitochondria. The concentration of carnitine and the rate of transfer of FFA into the mitochondria may limit the quantity of FFA used in aerobic energy production (Wenger and Reed, 1976; Holloszy, 1977). Endurance training leads to the overall enhancement of the aerobic potential of the cell. Increased oxidation of fat in the trained state could be due to the adaptive increase in muscle mitochondria and the increase in mitochondrial enzymes involved in the oxidation of FFA (Holloszy, 1977; Saltin et al., 1977).

At any given concentration of FFA, the rate of oxidation is highest in the muscles with the greatest capacity to oxidize fat. Heart muscle oxidizes fat more readily than slow oxidative muscle fibers (SO) and SO fibers more readily than fast glycolytic (FG) muscle fibers (Holloszy, 1977).

Work increases the rate of energy consumption and thus, increases the rate of energy production so that the rate of ATP formation can equal that of utilization (Newsholme, 1978; Wenger and Reed, 1976). The metabolic pathway producing ATP will depend on the intensity and duration of work. FG fibers rely primarily on anaerobic glycolysis for energy production whereas SO fibers rely on aerobic oxidation for energy supply. During high intensity short duration work, FG fibers are recruited. During low intensity long duration work, SO fibers are primarily recruited and produce energy by aerobic oxidation of fat, glucose and glycogen (Wenger and Reed, 1976).

The rate of FFA mobilization from adipose depots and FFA uptake by muscles are higher during exercise than at rest for any equivalent FFA level (McCafferty and Horvath, 1977). After an initial decrease in plasma FFA concentration, FFA levels increase during moderate submaximal exercise and also in recovery from exercise. Peak values in recovery occur after three to six minutes of rest (Hagenfeldt and Wahren, 1975).

Studies which measured RER show that both fat and carbohydrates are used jointly in different relative proportions by SO fibers (Christensen and Hansen, 1939; Astrand, 1977). The relative importance of fat or carbohydrate as a fuel is related to the intensity and duration of work. At rest and exercise of low intensity, long duration, FFA are the primary fuel used (Havel, 1971). Pruett (1970) showed that the direction of change in plasma FFA levels during work was dependent on the relative severity of work as measured by oxygen uptake. Well trained subjects exercising at 50 to 70% of $\dot{V}O_2$ max showed increasing levels of plasma FFA as exercise progressed. At 85 to 90% $\dot{V}O_2$ max, FFA concentration fell after the start of work and did not increase until recovery. FFA mobilization continued for several hours after exercise.

The relative amount of carbohydrate and fat utilized at different workloads also depends on the level of physical fitness with FFA oxidation being a more important source of energy in the trained than the untrained (Holloszy et al., 1971). The most marked difference between trained and untrained individuals in metabolic responses to the same relative work intensity is the proportion of fat and carbohydrate utilization. Trained subjects have lower plasma FFA concentrations both at rest and during exercise (Rennie et al., 1974; McCafferty and Horvath, 1977; Holloszy, 1977; Holloszy, 1978; Oscai, 1978; White et al., 1978; White et al., 1978, Bransford, 1979). The lower plasma FFA levels of the trained subjects during exercise could be related to either a depressed rate of lipolysis or to an elevated rate of uptake and utilization of FFA by the working muscle. Lower RER of trained subjects at similar relative loads as untrained subjects supports the concept of elevated extraction and oxidation following training (Christensen and Hansen, 1939; Holloszy, 1977; Saltin, 1977; Bransford, 1979). Sutton (1978) compared trained and untrained subjects working at identical absolute power outputs. The subjects worked at an absolute power outout of 750 kpm/min which was equal to 85% $\dot{V}O_2$ max for the untrained and 39.5% $\dot{V}O_2$ max for the trained. The fit group showed greater increases in FFA levels during exercise than the untrained subjects.

Exercise produces increased levels of lipolytic hormones - epinephrine, norepinephrine, cortisol, growth hormone, glucagon and adrenocorticotrophic hormone with a decrease in insulin levels (Pruett, 1971; Hartley et al., 1972; Bloom et al., 1976; Astrand, 1977; Sutton, 1978). Norepinephrine is the most powerful stimulator of FFA mobilization (Astrand, 1977) while growth hormone caused increased mobilization of FFA which lasted several hours during recovery from exercise (Pruett, 1971; Astrand, 1977).

Physical training is followed by a decrease in body fat and fat cell size (Bjorntorp, 1976) which implies that adipose tissue metabolism is altered towards increased mobilization and/or decreased lipid assimilation. Holm (1977) investigated the effect of exercise on fat cell metabolism. Fat cell biopsies were taken before and twenty-four hours after exercise at 70% $\dot{V}O_2$ max. Increased basal rates of lipid mobilization were found in the fat cells after physical exercise. These metabolic findings may show the initial changes in lipid metabolism produced by exercise which may lead to diminished fat depots.

Glucose

Resting blood glucose values vary from 69.38 to 108.12 mg % (Ahlborg et al., 1974; Wahren et al., 1971; Johnson and Rennie, 1973; Foster, 1978; Gollnick, 1978; Maehlum et al., 1978). Glucose levels determined using blood plasma are considerably higher (approximately 20 mg %) than glucose levels determined from whole blood (Foster, 1978). Glucose values are lower in the morning than the afternoon (Schlierf and Raetzer, 1972). Oral contraceptives do not effect resting glucose levels (Briggs, 1976). The greatest variability in plasma glucose levels of women are seen during the menstrual period (Southam and Gonzaga, 1965). The type and quantity of carbohydrate eaten affects resting glucose levels (Christensen and Hansen, 1939; Crapo et al., 1976; Thompson et al., 1978; Hultman, 1978).

Carbohydrates are stored in the body as glycogen in the liver (80-90 grams) and the muscle (300-400 grams) (Wahren, 1979). Muscle glycogen is used to meet the energy requirements of the muscle in which it is contained. Liver glycogen is a source of blood glucose which can be mobilized to maintain blood glucose homeostasis in response to exercise. The size of liver and muscle glycogen stores varies depending on diet. When a high carbohydrate diet is eaten after a period of a low carbohydrate diet, there is an increase in liver glycogen content (Hultman, 1978). Except for starvation in which the kidney produces glucose, the liver is the sole site of glucose production. At rest the rate of hepatic glucose production is 150 mg/min of which 75% is produced by glycogenolysis and the remainder by gluconeogenesis from lactate, pyruvate, glycerol and glucogenic amino acid (Wahren, 1977).

Glucose uptake by working muscle increases two to thirty-five times basal level with the increase being a function of both intensity and duration of work (Wahren, 1971; Ahlborg et al., 1974). The increase in glucose utilization by the muscle is met by increased splanchnic output of glucose. Exercise at 65W, 135W, 200W caused an increased splanchnic output which increased progressively with intensity to two, three and five times basal level after forty minutes of exercise (Wahren et al., 1974). The increased output was mainly the result of glycogenolysis. Gluconeogenesis decreases from 25-30% at rest to 15% after forty minutes of mild to moderate exercise to less than 5% after the same period of heavy exercise (Wahren, 1979). At low workloads for extended periods of time, the uptake of gluconeogenic precursors is of quantitative importance in maintaining glucose output (Hultman, 1978) by contributing to 45% of the overall hepatic glucose output (Wahren, 1979).

Blood glucose levels reflect the balance between splanchnic glucose output and its peripheral utilization. During exercise splanchnic glucose output increases to meet increased glucose uptake by the muscles. When exercise continues for an extended period of time, splanchnic glucose output fails to keep pace with peripheral glucose utilization resulting in decreased serum glucose levels. In order for an increase or decrease in serum glucose levels to be significant, it must be at least a change of 10 mg % (Udassin et al., 1977).

Changes in blood glucose levels during exercise depend upon intensity and duration of exercise as well as the fitness level of the subject. Wahren (1971) measured the glucose concentration during exercise of forty minutes duration at 400, 800 or 1200 kpm/min on subjects who were not training regularly. Blood glucose levels did not significantly change during mild exercise at 400 kpm/min. Workloads of 800 and 1200 kpm/min elicited a gradual rise in glucose concentration which was more pronounced at the heaviest workloads. Most researchers have found similar results of no change in blood glucose levels of untrained individuals during moderate intensity exercise (Rottini, 1971; Johnson and Rennie, 1973; Udassin, 1977; White et al., 1978; Sutton, 1978). Bloom (1976) found blood glucose to increase in untrained subjects at 30% $\dot{V}O_2$ max. Felig and Wahren (1979) showed a significant increase in blood glucose levels of untrained subjects during exercise of 55-65% $\dot{V}O_2$ max. Pruett (1970) found blood glucose levels to decrease at 50-70% of $\dot{V}O_2$ max in trained subjects. During high intensity work, most researchers have found blood glucose levels to rise (Pruett, 1970; Wahren, 1971; Bloom et al., 1976; White et al., 1978; Sutton, 1978). The increase was found to be greater in trained than untrained subjects (Bloom et al., 1976; White et al., 1978).

Plasma insulin levels have been observed to decrease during acute exercise and heavy, prolonged exercise in spite of increased glucose utilization (White et al., 1978). In the complete absence of insulin, glucose uptake does not increase during work (Vranic and Wrenshall, 1969) suggesting that insulin may exert a permissive effect on exercise induced glucose uptake (Wahren, 1979). The hormonal response to the end of work is an increase in insulin levels within 2 to 10 minutes (Pruett, 1971; Felig and Wahren, 1975).

Exercise causes an increase in plasma glucagon. Glucagon may inhibit insulin secretion and increase substrates for exercising muscles by increased glycogenolysis and lipolysis (Wahren, 1971; Luyckx et al., 1978). Increases in glucagon are more pronounced when exercise is prolonged or severe (Wahren, 1979). Glucagon remains high after exercise and may contribute to increased hepatic uptake of gluconeogenic precursors (Felig and Wahren, 1975). Glucagon and insulin play an important role in glucose homeostasis. Glucagon appears to be mainly responsible for hepatic glucose output while insulin regulates peripheral glucose uptake.

Catecholamines indirectly affect glucose uptake. An increase in catecholamines results in increased FFA mobilization and increased glycogenolysis which both decrease glucose utilization (Vranic and Berger, 1979). Issekutz (1979) blocked the effect of catecholamines in exercising dogs which produced a decreased turnover of FFA and decreased glycogenolysis. This resulted in increased glucose utilization by the muscle.

Increased elevation of plasma glucose and insulin prior to exercise result in increased carbohydrate metabolism during exercise (Costill, 1977). The type and quantity of carbohydrate eaten determine resting glucose and insulin levels (Christensen and Hansen, 1939; Pruett, 1971; Reaven and Olefsky, 1974; Crapo et al., 1976; Hultman, 1978; Thompson et al., 1978). Thompson et al. (1978) showed that sucrose containing diets produced significantly lower insulin concentrations than corn syrup containing diets. A low fat, high carbohydrate diet resulted in increased resting glucose and insulin levels (Reaven and Olefsky, 1974; Maughan et al., 1978). The insulin levels were elevated out of proportion to the rises in plasma glucose concentrations. After a high carbohydrate diet, blood glucose increased more during recovery than on a normal mixed diet (Maughan et al., 1978). High fat, low carbohydrate diets result in decreased resting insulin and glucose levels (Pruett, 1971) which increase the oxidation of FFA during exercise (Christensen and Hansen, 1939). The oxidation of lipids can never fully replace the use of glucose by contracting muscles even in trained individuals.

Lactate

The relative intensity of work dictates the change of plasma FFA levels as well as the amount of lactate formed (Hermansen, 1971; Pruett, 1970; Wenger and Reed, 1976). High lactate levels inhibit FFA mobilization during exercise (Miller et al., 1963; Issekutz et al., 1966; Boyd, 1974).

Issekutz and Miller (1962) found an inverse relationship between plasma FFA levels and lactate production which indicated that lactate may function as a physiological inhibitor of FFA mobilization during severe exercise. Subsequent studies showed that decreased FFA levels resulted from increased lactate levels which inhibited fat mobilization from adipose tissue while FFA uptake continued in the exercising muscle (Miller et al., 1963; Issekutz et al., 1966). Although FFA release from adipose tissue is inhibited by high lactate concentrations, glycerol release is stimulated (Dieterle, 1971) indicating that lactate may cause increased re-esterification of FFA (Astrand, 1977). Boyd (1974) found that lactate concentrations of 6 to 8 mM/1 produced decreased FFA levels. Thus, there is a critical lactate concentration which when exceeded will inhibit fat mobilization and decrease FFA levels.

The rate of energy production during exercise determines the metabolic pathway used to produce energy which in turn determines the formation of lactate. During high intensity exercise of short duration, lactate is formed primarily in FG fibers by anaerobic glycolysis (Fox and Mathews, 1976). FG fibers contain high concentrations of LDH-M which facilitates the formation of lactate from pyruvate (Wenger and Reed, 1976; McGrail, 1977). Lactate diffuses from the muscle to accumulate in the blood (McGrail et al., 1977). During light to moderate intensity work, energy production is met almost exclusively by the aerobic oxidation of glucose or FFA in SO fibers and lactate levels remain near resting levels (Hermansen, 1971).

The intensity of exercise at which lactate production increases depends on the type of activity as well as the participant's fitness level. Karlsson (1971) found a marked increase in lactate production at 50-60% of $\dot{V}O_2$ max in relatively fit subjects. Pruett (1970) found that lactate levels remained low in well trained athletes at 70% $\dot{V}O_2$ max. At increasing intensities of 80-90% $\dot{V}O_2$ max, lactate levels were elevated. Bloom et al. (1976) found mild exercise of 30% $\dot{V}O_2$ max to cause a significant rise in lactate levels of untrained subjects. Brandsford and Howley (1979) studied untrained women and found that lactate levels remained at or slightly above resting levels during exercise which required 55% of $\dot{V}O_2$ max. As exercise becomes more severe in relation to the subject's $\dot{V}O_2$ max, a larger proportion of energy is derived anaerobically. The mobilization of FFA is depressed as long as blood lactate levels remain high.

Blood lactate concentrations are lower in trained than untrained subjects working at the same absolute workload (Sutton, 1978) and at the same relative workload (Cobb and Johnson, 1963; Hermansen, 1971; Hartley et al., 1972; Rennie et al., 1974; Bloom et al., 1976). After training, reduced blood lactate concentrations at a given submaximal workload result because the muscles have adapted to training with an increased respiratory capacity (Saltin and Karlsson, 1971; McGrail et al., 1977; Holloszy, 1978). A higher intensity is needed to attain the same rate of lactate production in the trained as compared to the untrained muscle. Saltin and Karlsson (1971) found that twelve weeks of endurance training resulted in significant decreases in lactate at a given submaximal workload and that after twenty-eight weeks of training lactate production was significantly reduced at the same relative workload. It can be seen that intensity of exercise is an important factor in a weight reduction training program as it affects both FFA mobilization and lactate formation.

CHAPTER III

METHODOLOGY

Subject Selection and Orientation

Fifty female volunteer subjects were screened to meet the following requirements: 25% or greater body fat, weight stable for at least two months, healthy sedentary person not involved in regular physical activity, non-smokers, not currently using medications other than oral contraceptives, less than thirty years old. During the screening procedures, subjects were required to complete a questionnaire (Appendix A), were hydrostatically weighed and were orientated to the procedures involved in stress testing (i.e., pedalling a Monarck bicycle ergometer at increasing workloads while breathing through a rubber mouthpiece attached to a Rudolph valve). Subjects were required to sign an informed consent form which was approved by the faculty ethics committee (Appendix I).

All subjects did not meet the above criteria. However, thirty-four subjects who best met the criteria were selected so that there would be enough subjects for each group. They ranged in age from nineteen to thirty-four years ($\overline{X}$ = 23.9 years SD = 3.4 years). During the study, subjects were asked to refrain from any regular vigorous physical activity other than that associated with the experiment and to maintain their usual eating patterns.

Testing Procedures

Maximal Stress Test

$\dot{V}O_2$ max for each subject was determined using a Quinton electric bicycle ergometer. Prior to each test, subjects clad in T-shirts and shorts were weighed on a balance scale. Heart rate and electrocardiogram (ECG) patterns were monitored on a Sanborn 500 Viso-Cardiette. A qualified cardiac nurse was present to interpret the ECG patterns and to establish maximum heart rate. Subjects were fitted with a nose clip and a rubber mouthpiece attached to a Rudolph valve which enabled measurement and analysis of expired gases by a Metabolic Measurement Cart (Beckman Instruments, Inc., Illinois). Calibration of this instrument was conducted before and after each test.

A four minute warm up was performed at a power output of 200 or 400 kpm/minute depending on body size and previous activity level of the subject. Speed of cycling was maintained at 60 revolutions per minute. Power output was increased by 200 kpm/min at the beginning of the fifth minute, again at the seventh minute and then by 100 kpm/min at the ninth minute and every minute thereafter until volitional exhaustion or $\dot{V}O_2$ max had been reached.

The criteria used to determine $\dot{V}O_2$ max was a levelling off of $\dot{V}O_2$ max as indicated by less than 150 ml/min increase in $\dot{V}O_2$ over the previous power output (Taylor, 1955).

Hydrostatic Weighing

A rectangular tank six feet in height, four feet in width, and ten feet in length was used for hydrostatic weighing. Within the tank, an aluminum chair was suspended from a load cell which was connected to a

Sargent recorder (model SR). Prior to each test, the recorder was calibrated and water temperature was measured and recorded. Temperature of the room was not controlled but remained within a range of 28°C to 32°C.

Hydrostatic weighing sessions were conducted in the morning with subjects in a postabsorptive state after fasting for twelve hours. Before entering the tank, subjects dressed in swim suits and were weighed on a balance scale to the nearest one quarter of a pound. Upon entering the tank, subjects were seated in the chair and a twenty pound diver's weight belt was placed across the thighs. Vital capacity was measured using a Collins 9 litre spirometer with the subject seated in the water to neck level. The largest volume of three trials was assumed to be the best estimate of vital capacity.

The technique for hydrostatic weighing was as follows:

1. air bubbles were dislodged from hair and body
2. the subject maximally inhaled and closed the nasal passage
3. subject leaned forward from the waist until the body was completely submerged.

This procedure was repeated until two similar chart readings were obtained; the recording that indicated the greatest inhalation was used as the hydrostatic weight (Moyer, 1971).

Residual volume was estimated as 25% of the vital capacity (Comroe, 1966). Per cent body fat was calculated according to the formula of Brozek (Brozek et al., 1963).

Subject Grouping for Blood Metabolite Measurements

Blood samples were taken pre and post exercise during the first and last training sessions of the high intensity exercise group and the low intensity exercise group. Each control subject was randomly assigned to a high or low intensity exercise session. Three days later, each control subject completed the second workout of opposite intensity so that all control subjects did both a high and low intensity exercise session.

Blood Sampling Procedure

Blood samples were taken to determine pre and post exercise values for free fatty acids, glucose and lactate. Subjects reported to the laboratory after fasting for twelve hours. Blood samples were taken at rest and after three minutes of recovery from exercise. Approximately 5 ml of blood were drawn by a qualified technician using a 22 guage venoject blood collecting needle (Terumo) with a heparinized vacutainer. 0.2 ml of blood were immediately pipetted into 7% perchloric acid and 0.1 ml of blood were pipetted into 4% perchloric acid. Blood samples were refrigerated until test completion at which time all samples were centrifuged at 3600 rpm for ten minutes. Samples were frozen at -70.0°C until analysed. A colorimetric method was used to assay the serum for FFA (Falholt, Lund, Falholt, 1973). Lactate concentrations were determined by a spectrophotometry assay (Mohme-Lundholm, 1965). Glucose levels were determined by the assay of Bergmeyer (Bergmeyer, 1974). All samples were analyzed in triplicate. A Unicam SP 1800 Ultraviolet spectrophotometer was used to measure optical densities.

Assignments of Subjects to Training Groups

Following pre-training tests, subjects were ranked according to per cent body fat, age and $\dot{V}O_2$ max and then were randomly assigned to one of the following:

1. high intensity exercise program designed to maintain 80% of pre training maximum heart rate (N = 12)
2. low intensity exercise program designed to maintain 65% of pre training maximum heart rate (N = 12).
3. control group instructed to maintain their same diet and activity pattern during the training period (N = 10).

Training Procedure

The training program consisted of pedalling a Monarck bicycle ergometer three times per week on alternate days for nine weeks. A warm up consisted of pedalling for three minutes at 50% of maximum heart rate for the low intensity group and 65% of maximum heart rate for the high intensity group. Workloads were adjusted to produce either 65% or 80% of maximum heart rate for the duration of each session. The time involved in each session depended on a predetermined total power output. Total power output was set at 12,000 kpm for the first four weeks and then was increased to 14,000 kpm for the remainder of the program. Thus the length of each individual's training session depended on the intensity of exercise and their physical condition with times generally varying from twenty to forty minutes. Subjects pedalled at a rate of 60 rpm and heart rates were monitored on a Cardiometer (Cardionics ab. Stockholm, Sweden). Cardiometers and bicycle ergometers were calibrated on a weekly basis.

Diet and Exercise Records

In order to determine if usual dietary and exercise patterns were maintained, subjects were instructed to fill out a one week diet record and a three day exercise record prior to the commencement of the study and twice during the study. When discrepancies were noted, personal interviews were conducted to further establish dieting patterns.

Statistical Analysis

The statistical analysis of the results involved a two way analysis of variance on repeated measures for % fat, pounds fat, fat free weight, body weight, $\dot{V}O_2$ max and a three-way analysis of variance with repeated measures on the last factor for the blood parameters of FFA, glucose and lactate. The statistical computations were made using ANOV 23 and ANOV 30 (DERS programs). Post hoc procedure, where appropriate, were performed using a Scheffé test for determining significant mean differences. Significant differences were accepted at the alpha level p is less than 0.05 where p is the probability that no difference exists between means.

Experimental Design

The study consisted of two parts:

1) the effect of training on body composition, and
2) training effects on blood metabolites.

The experimental design for the effect of training on body composition was a 2 X 3 factorial design with repeated measures on the last factor; the independent variable was intensity of exercise. The dependent variables of the study were changes in body composition, body weight and $\dot{V}O_2$ max. A diagrammatical representation of the design is presented in Table 1.

The second part of the study investigated the effect of training on resting and post exercise levels of free fatty acids, glucose and lactate. The experimental design was a 2 X 2 X 2 factorial design with repeated measures on the last factor. Factor A was the group of subjects (exercise, control). Factor B was intensity of exercise (high, low). Factor C was the repeated measure on the following:

(a) pre-pre - pre training resting values
(b) pre-post - pre training post exercise values
(c) post-pre - post training resting values
(d) post-post - post training post exercise values.

The dependent variables were FFA, glucose and lactate levels. Table 2 represents a diagrammatical description of the design.

TABLE 1

EXPERIMENTAL DESIGN OF THE TRAINING PROGRAM

	Pre Tests	Treatment 9 Weeks	Post Tests
Group 1	X	High Intensity Training	X
Group 2	X	Low Intensity Training	X
Group 3	X	No Training	X

TABLE 2

EXPERIMENTAL DESIGN FOR BLOOD PARAMETERS

	Trials			
	Pre-Training		Post Training	
	Pre-Exercise	Post Exercise	Pre-Exercise	Post Exercise
Exercise A_1		B_1 (High Intensity)		C_1 (pre-pre) (pre-post)
		B_2 (Low Intensity)		C_2 (post-pre) (post-post)
Control A_2		B_1 (High Intensity)		C_1 (pre-pre) (pre-post)
		B_2 (Low Intensity)		C_2 (post-pre) (post-post)

CHAPTER IV

RESULTS

Complete data were collected on thirty of thirty-four subjects who participated in the study. The characteristics of these subjects are described in Table 3. No statistical difference ($p. > 0.05$) was shown to exist between control and exercise groups on pre training percent body fat, $\dot{V}O_2$ max and age.

The effects of training on body composition and $\dot{V}O_2$ max are described in Table 4. A two-way analysis of variance showed no significant ($p > 0.05$) "A" main effects on percent body fat, pounds fat, fat free weight or $\dot{V}O_2$ max indicating that there were no differences between groups. Body weight showed a significant "A" main effect ($p < 0.05$).

TABLE 3

CHARACTERISTICS OF THE SUBJECTS

		High Intensity N=10	Low Intensity N=10	Control N=10
Weight (lb)	$\bar{X} \pm SD$ Range	127.70 + 9.89 (111.00 - 140.50)	124.10 + 12.73 (110.25 - 145.50)	138.53 + 17.37 (112.50 - 173.00)
VO_2 max (ml/kg/min)	$\bar{X} \pm SD$ Range	36.92 + 5.15 (31.20 - 43.10)	36.75 + 4.59 (30.00 - 43.50)	36.56 + 4.83 (29.70 to 44.70)
Age (in years)	$\bar{X} \pm SD$ Range	24.90 + 3.73 (21 - 32)	24.60 + 3.47 (18 - 31)	22.20 + 2.53 (18 - 26)
% Fat	$\bar{X} \pm SD$ Range	27.39 + 3.60 (21.53 - 34.19)	27.22 + 3.19 (20.37 - 32.61)	27.80 + 5.8 (19.79 - 37.33)

TABLE 4

THE EFFECT OF TRAINING ON BODY COMPOSITION AND $\dot{V}O_2$ MAX*

	High Intensity		Low Intensity		Control	
	Pre	Post	Pre	Post	Pre	Post
% Fat	27.39± 3.60	25.44± 2.66	27.22± 3.19	25.29± 3.32	27.80± 5.80	28.11± 5.01
lbs Fat	35.07± 5.71	32.63± 4.27	34.12± 6.17	31.22± 6.83	38.44± 10.43	40.15± 11.49
Fat Free Weight (lbs)	92.71+ 7.66	95.48± 7.63	91.54± 9.04	94.91± 8.92	99.63± 10.80	99.96± 10.40
Body Weight (lbs)	127.7± 9.89	128.20± 9.47	124.10± 12.73	124.41± 13.28	138.53± 17.37	139.56± 16.51
$\dot{V}O_2$ max (ml/kg/min)	36.92± 5.15	39.51± 2.13	36.75± 4.59	38.51± 3.89	36.56± 4.83	34.05± 4.16

* Values represent $\bar{X} \pm$ SD

Significance ($p < 0.05$) was shown, however for "B" main effect (treatment effect) and "AB" interactions (different slopes of improvement) (Appendices B-1, B-2, B-5, B-7, B-9).

After training, the high intensity exercise group showed a decrease ($p < 0.05$) in percent body fat from 27.39% to 25.44%, a change of 1.95% (Figure 1). The low intensity exercise group showed a decrease in the average percent body fat from 27.22% to 25.29% ($p < 0.05$), a change of 1.93%. The control group showed a non-significant increase in percent body fat from 27.8% to 28.11% ($p > 0.05$) (Appendix B-2).

From pre to post training, the low intensity exercise group showed an average decrease of 2.9 pounds of fat ($p < 0.05$); the high intensity exercise group showed an average decrease of 2.44 pounds of fat

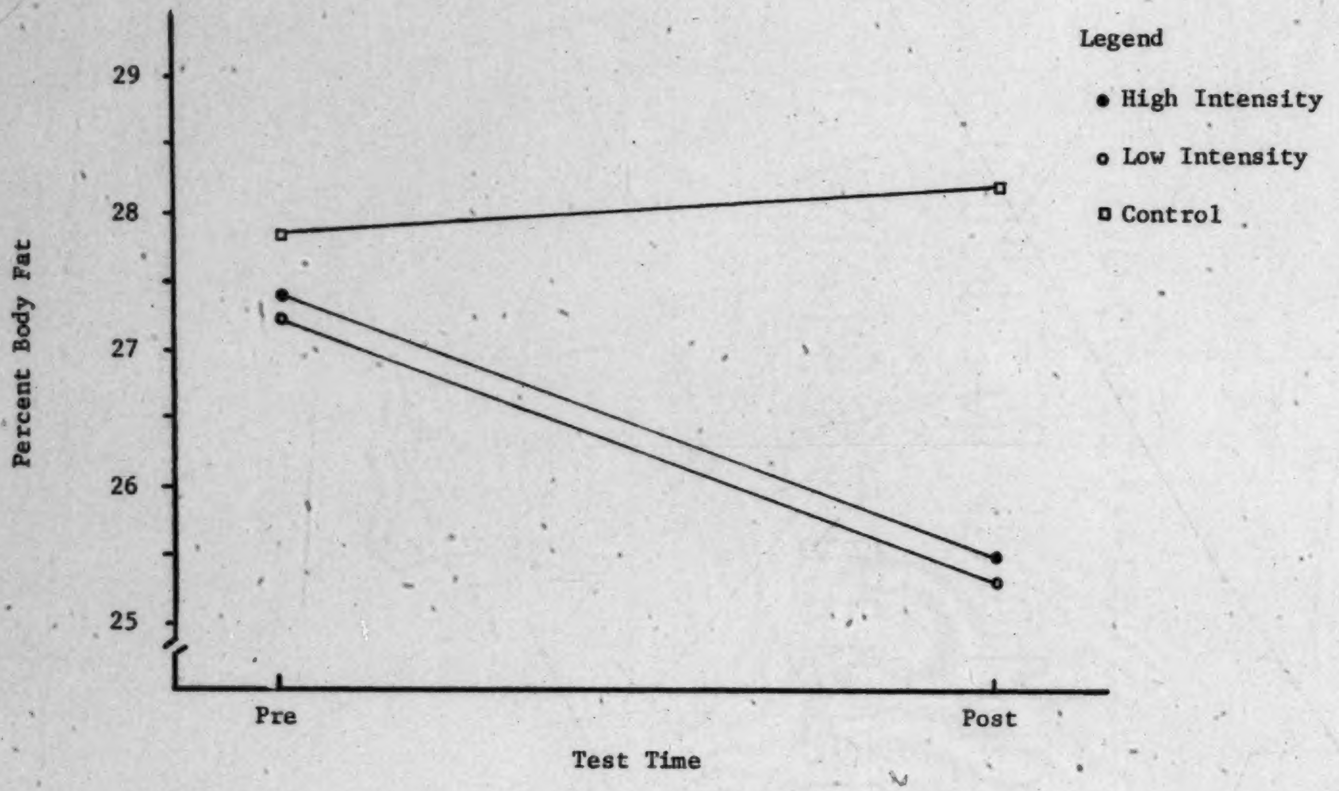

FIGURE 1. THE EFFECTS OF TRAINING ON PERCENT BODY FAT

($p > 0.05$); and the control group showed an average increase ($p > 0.05$) of 1.71 pounds of fat (Figure 2). Both the high and low intensity exercise groups had fewer pounds of fat than the control group prior to and following training ($p < 0.05$) (Appendix B-4).

The high intensity exercise group gained 2.78 pounds of fat free weight after training ($p < 0.05$); the low intensity exercise group gained 3.37 pounds of fat free weight ($p < 0.05$) and the control group showed no change ($p > 0.05$) (Figure 3). Both the high and low intensity exercise groups had lower fat free weights than the control group prior to and following training ($p < 0.05$) (Appendix B-6).

No decrease in body weight occurred in any of the groups ($p > 0.05$) (Figure 4). A two-way analysis of variance showed an "A" main effect between the groups ($p < 0.05$) (Appendix B-7). However, when post hoc comparisons were made using the Scheffé procedure no significant differences in body weight between groups were obtained (Appendix B-8).

There were no significant increases ($p > 0.05$) in $\dot{V}O_2$ max for any of the groups (Figure 5). However, post training values for both the high and low intensity exercise groups were greater than those of the control group ($p < 0.05$) (Appendix B-10).

A three-way analysis of variance was used to determine the effect of training on resting and post exercise lactate values (Appendix C-1, C-2). The results are summarized in Table 5.

There were no significant differences ($p > 0.05$) between high and low intensity exercise or control groups for resting levels of lactate pre and post training (Figure 6).

The high intensity groups produced a greater amount of lactate after exercise than the low intensity groups both pre and post training

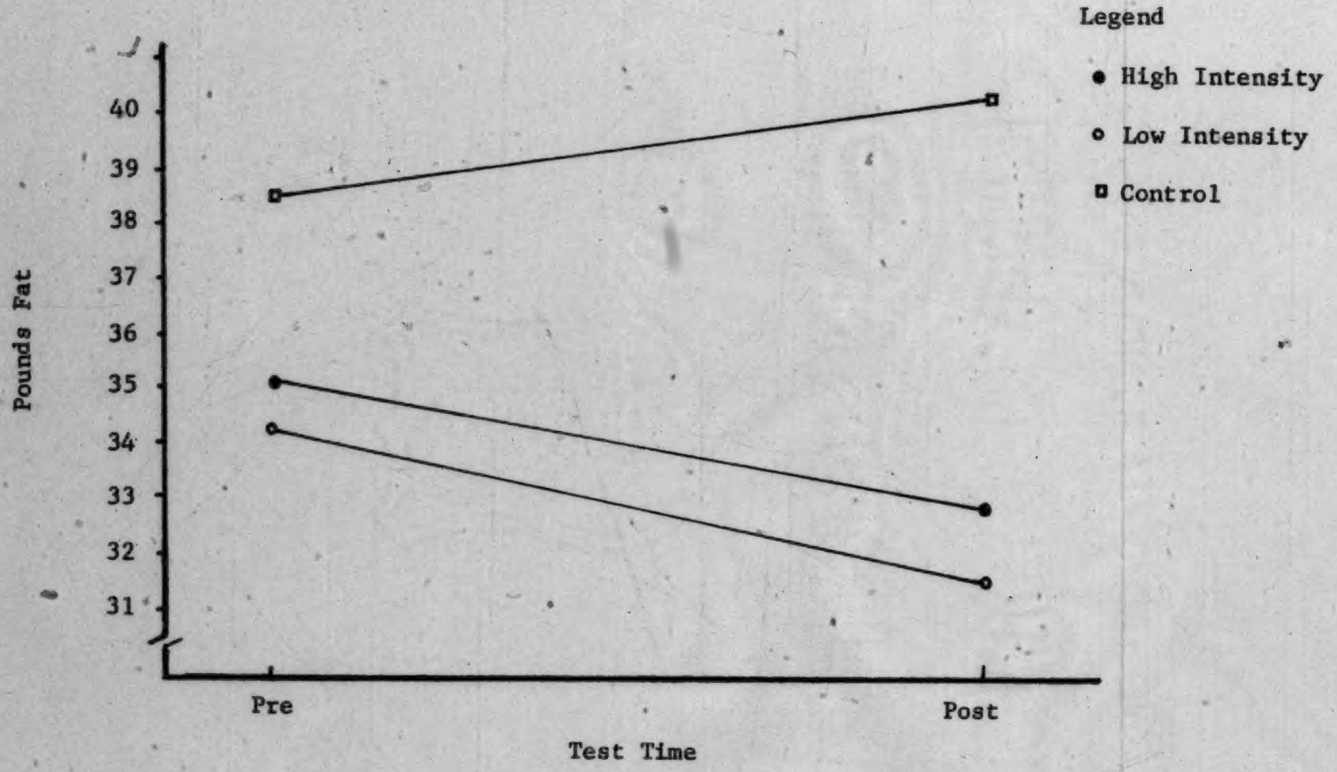

FIGURE 2. THE EFFECTS OF TRAINING ON POUNDS FAT

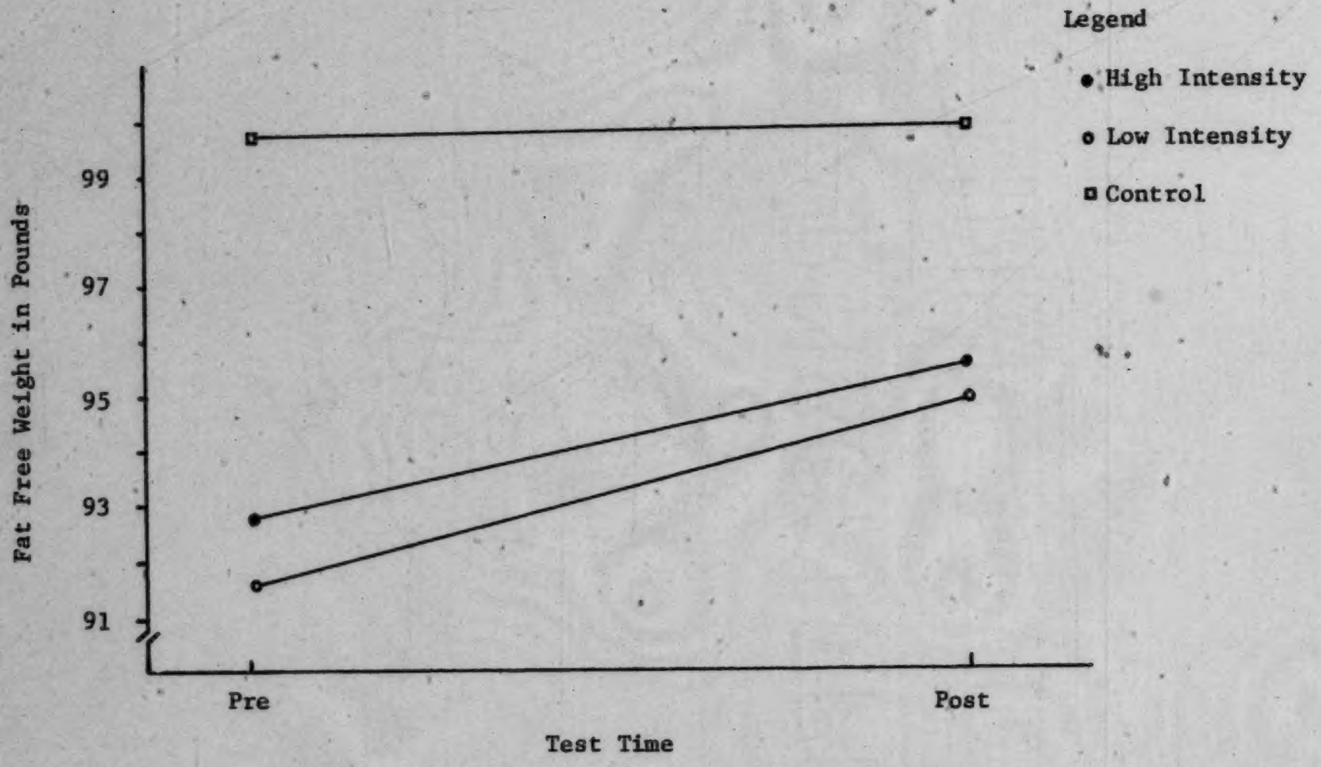

FIGURE 3. THE EFFECTS OF TRAINING ON FAT FREE WEIGHT

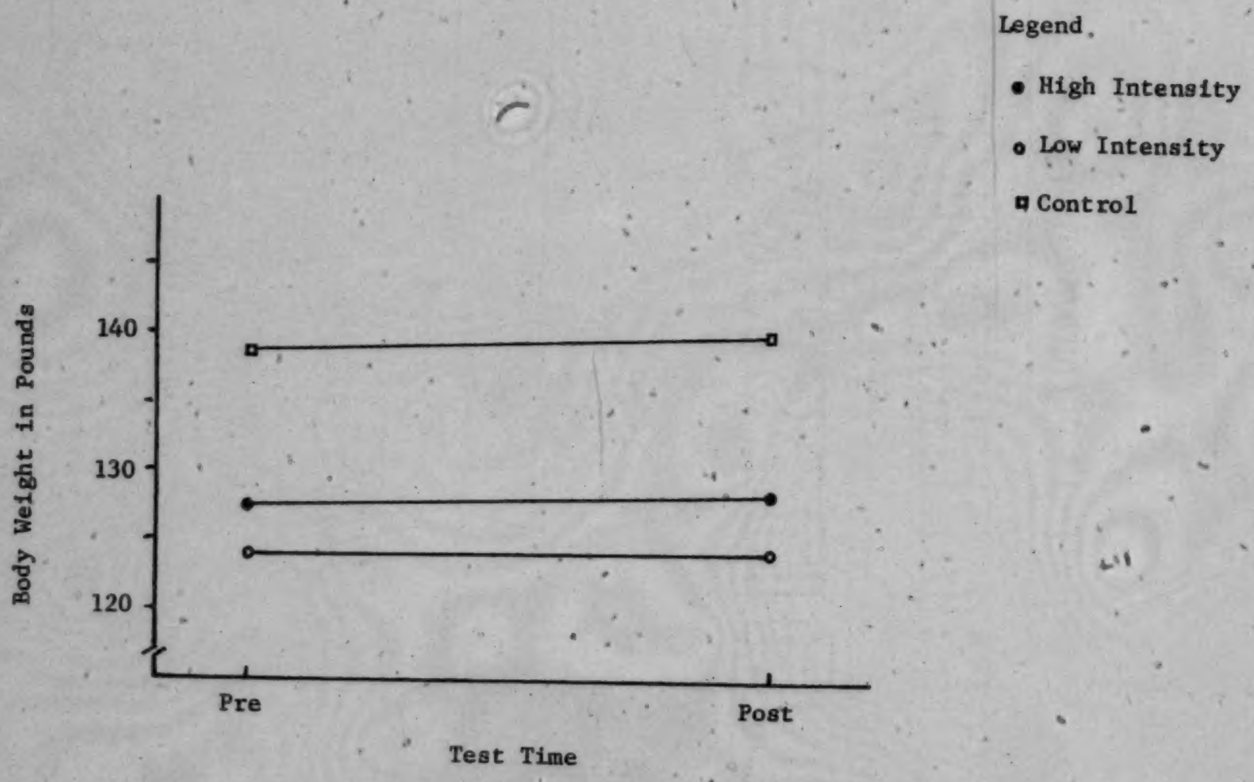

FIGURE 4. THE EFFECTS OF TRAINING ON BODY WEIGHT

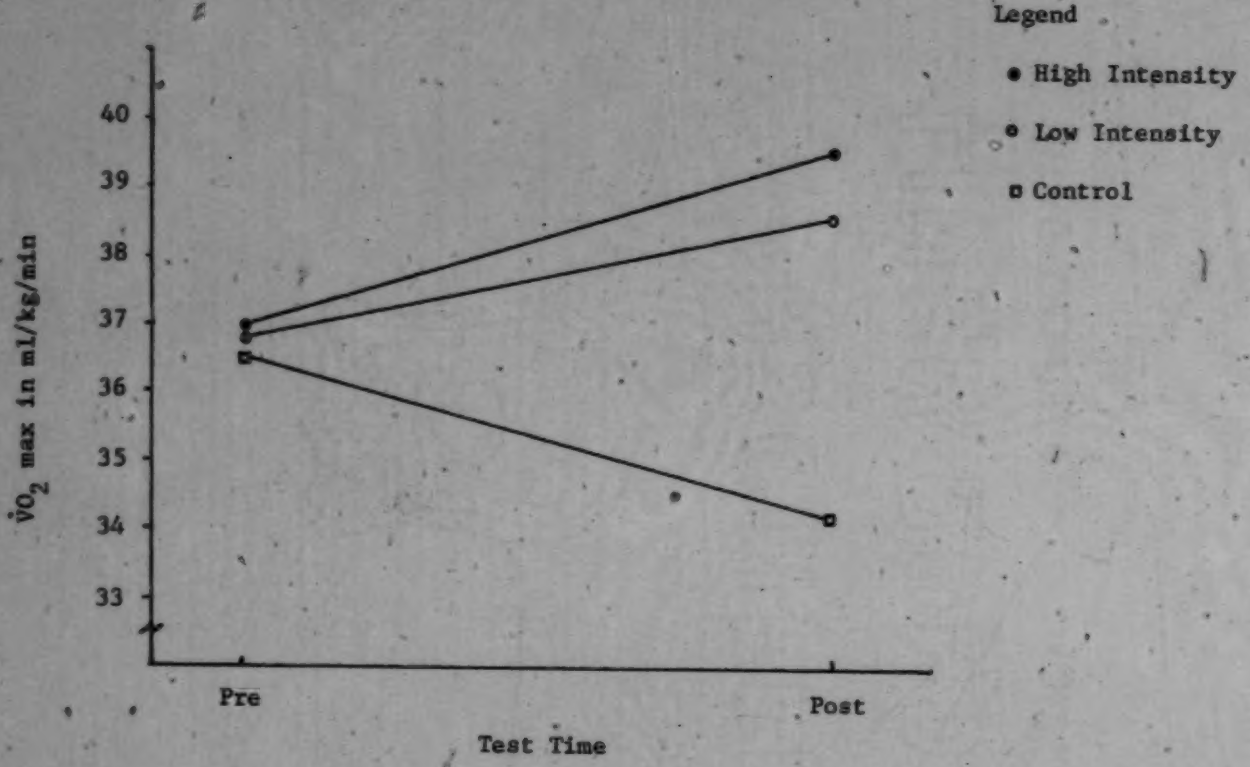

FIGURE 5. THE EFFECTS OF TRAINING ON $\dot{V}O_2$ MAX

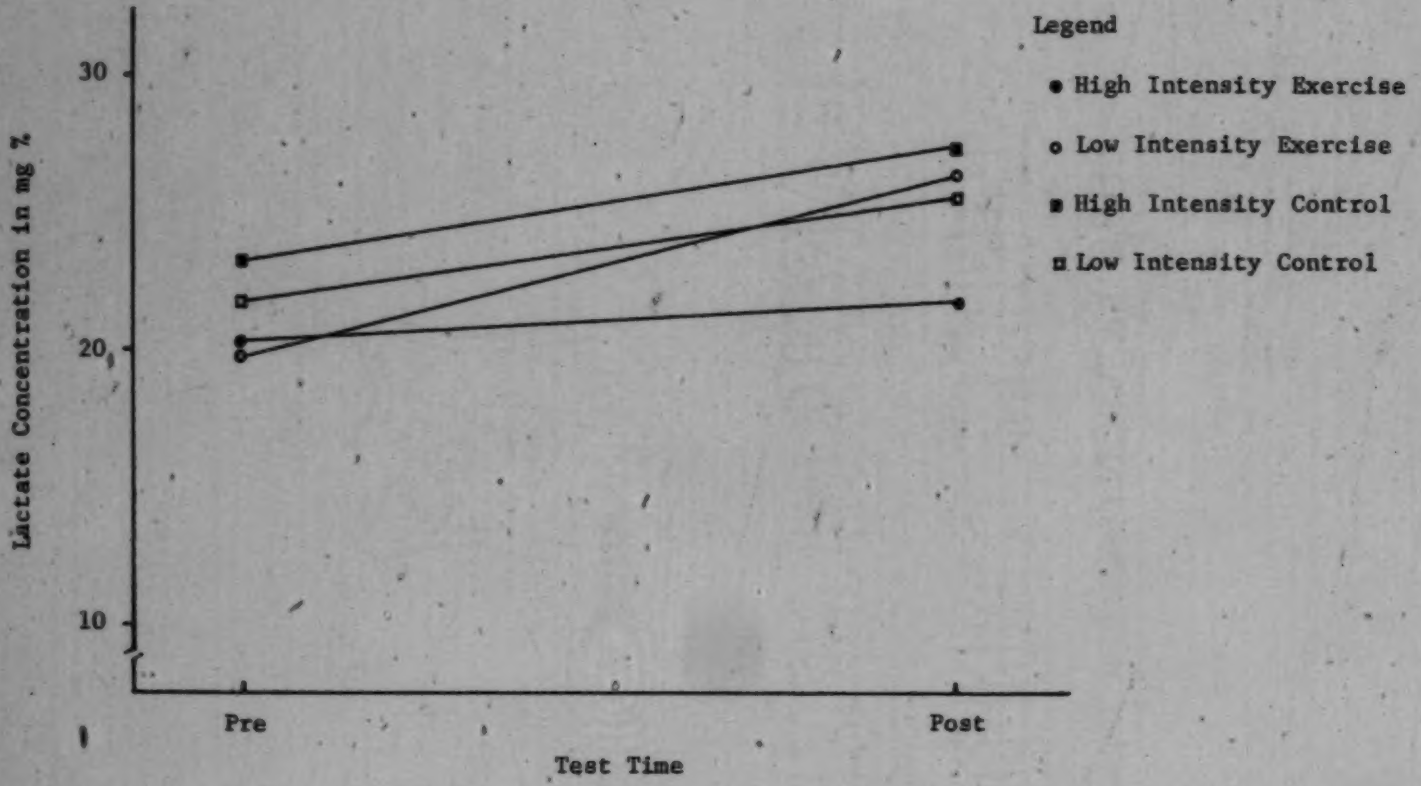

FIGURE 6. THE EFFECTS OF TRAINING ON RESTING LACTATE CONCENTRATIONS

TABLE 5

THE EFFECT OF TRAINING ON RESTING AND POST EXERCISE MEAN LACTATE LEVELS IN mg %*

		Resting Values		Post Exercise Values	
		Pre Training	Post Training	Pre Training	Post Training
Exercise	High Intensity	20.72 ± 7.75	21.48 ± 15.73	37.76 ± 9.01	36.69 ± 12.33
	Low Intensity	19.91 ± 5.66	26.25 ± 4.89	32.47 ± 13.60	31.48 ± 6.74
Control	High Intensity	23.30 ± 8.15	27.22 ± 9.12	45.47 ± 7.73	55.74 ± 16.26
	Low Intensity	21.74 ± 6.29	25.18 ± 9.28	34.39 ± 8.91	37.44 ± 13.37

* Values $\overline{X}$ ± SD

($p < 0.05$) (Figure 7). The high intensity control group had significantly higher concentrations of lactate than the low intensity control group after exercise both pre and post training ($p < 0.05$). The high intensity and low intensity exercise groups followed the same trend ($p > 0.05$) (Appendix C-3).

The high intensity control group produced more lactate ($p < 0.05$) than the high intensity exercise group both pre and post training (Figure 7). There were no differences ($p > 0.05$) between exercise and control groups for post lactate exercise values prior to training (Figure 7). Following training, the control groups produced more lactate than the exercise groups ($p < 0.05$).

A three-way analysis of variance was used to determine the effect of training on resting and post exercise FFA values. The results are summarized in Table 6.

The effect of training on resting FFA levels yielded a significant "C" main effect (Appendix C-4) indicating a decrease ($p < 0.05$) in resting FFA levels from pre to post training for the exercise groups while no difference ($p > 0.05$) occurred in the control groups (Figure 8). There was no difference ($p > 0.05$) between high and low exercise groups in the decrease of resting FFA levels (Appendix C-5).

Plasma concentrations of FFA were higher in the exercise groups than the control groups (Table 6) following the initial exercise session ($p < 0.05$). There was no difference ($p > 0.05$) between high and low intensity groups in the amount of FFA mobilized after exercise (Figure 9). The exercise groups showed lower concentrations of FFA after the final exercise session ($p < 0.05$) while the controls had concentrations similar to pre training values ($p > 0.05$). The low

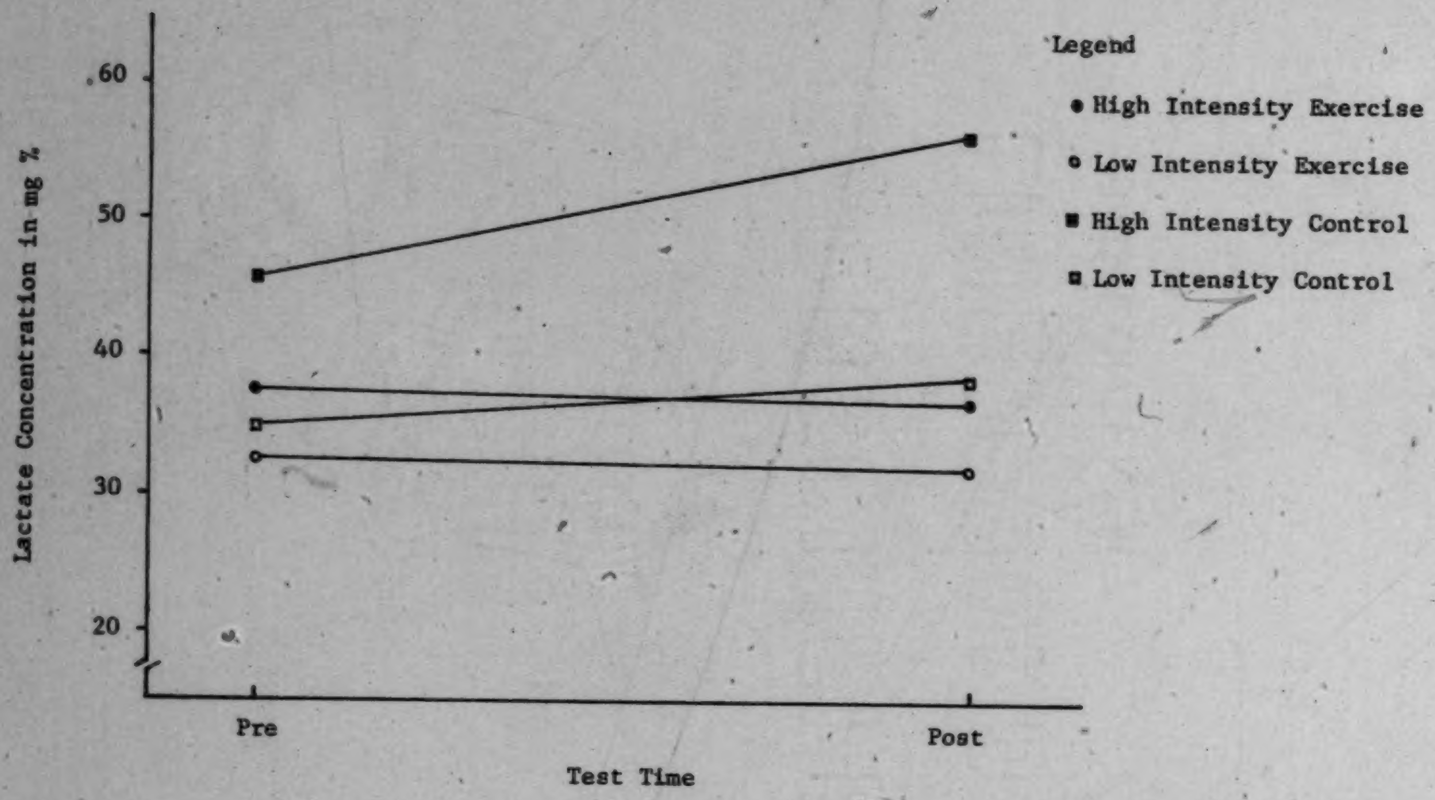

FIGURE 7. THE EFFECTS OF TRAINING ON POST EXERCISE LACTATE CONCENTRATIONS

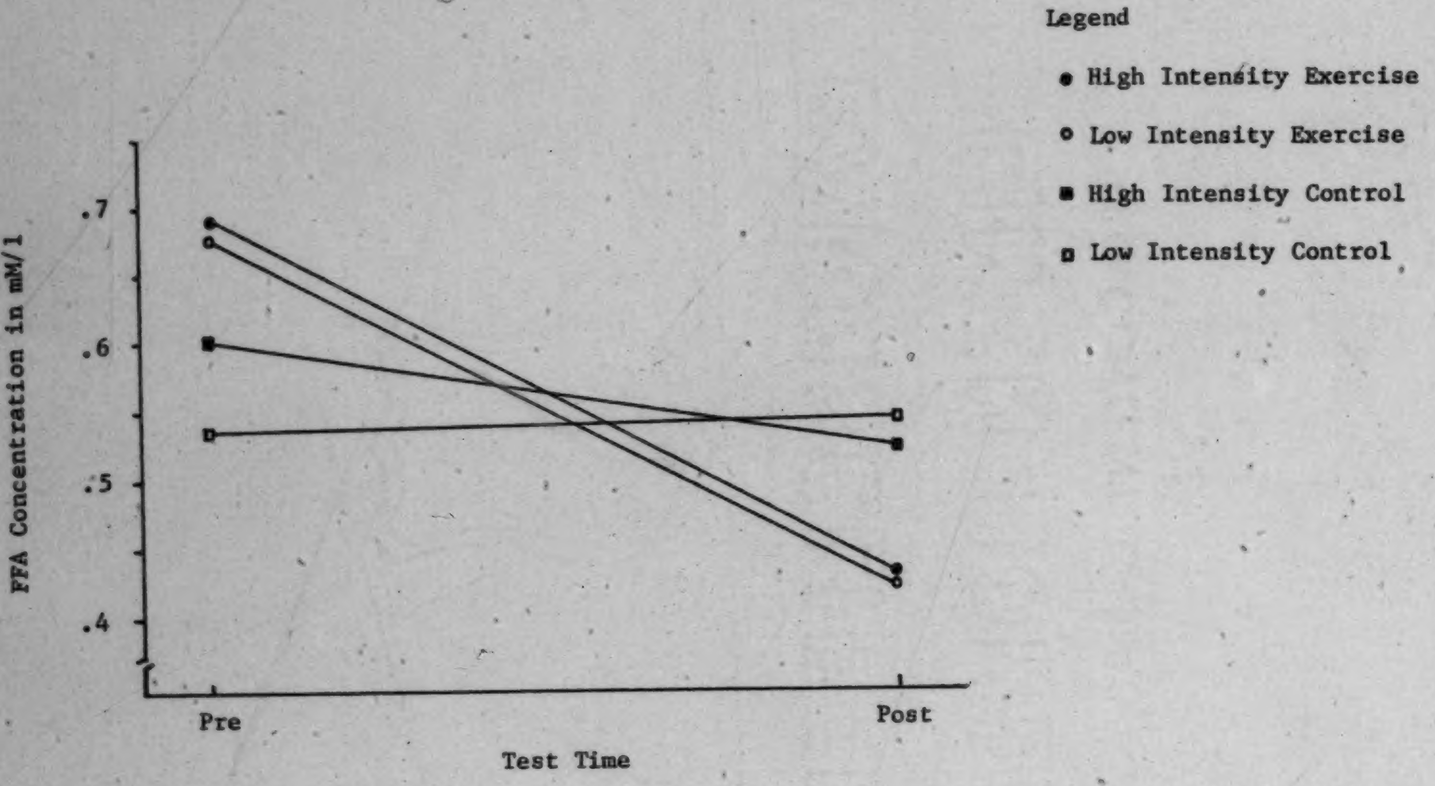

FIGURE 8. THE EFFECTS OF TRAINING ON RESTING FFA CONCENTRATIONS

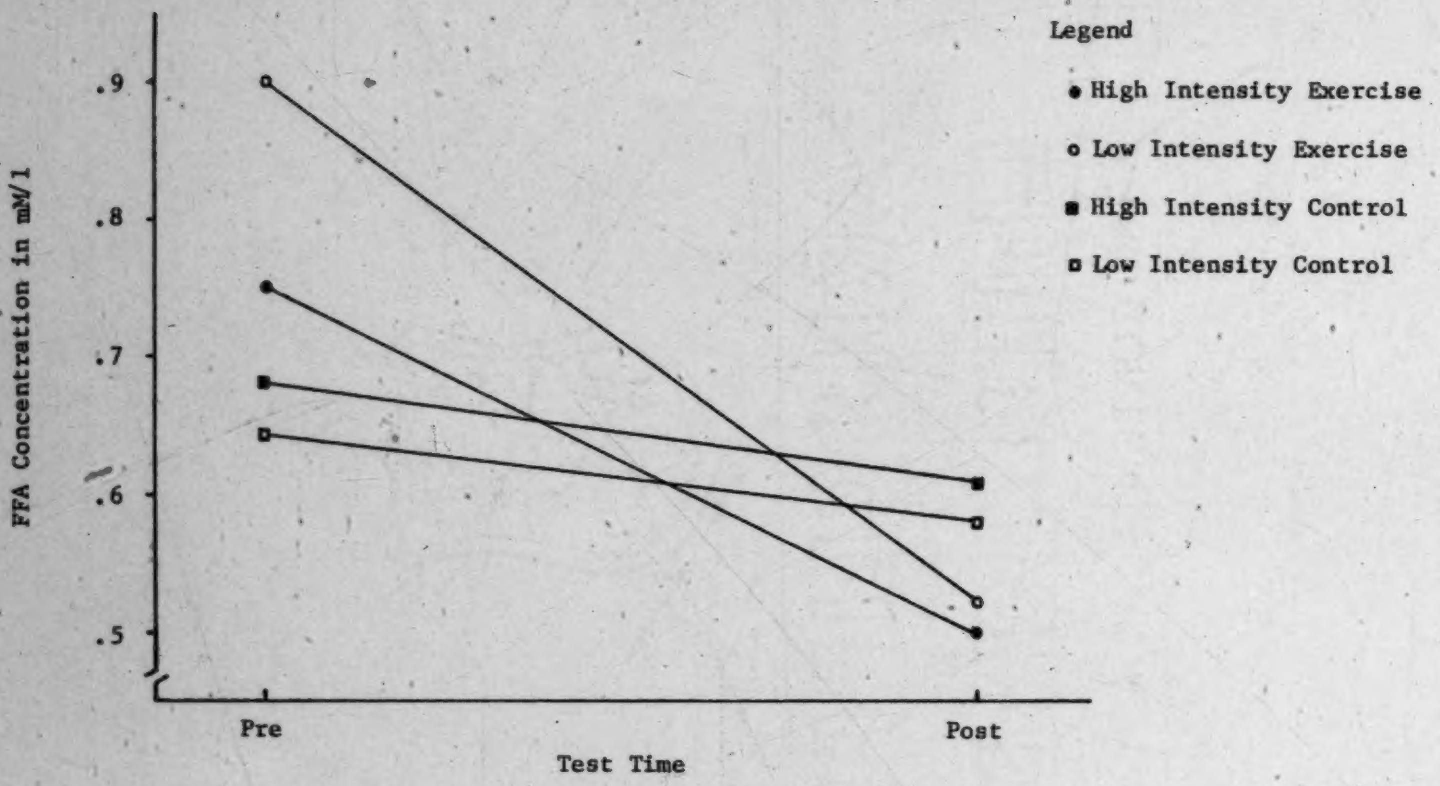

FIGURE 9. THE EFFECTS OF TRAINING ON POST EXERCISE FFA CONCENTRATIONS

TABLE 6

THE EFFECT OF TRAINING ON RESTING AND POST EXERCISE PLASMA FFA LEVELS IN mM/1*

		Resting		Post Exercise	
		Pre Training	Post Training	Pre Training	Post Training
Exercise	High Intensity	.686 ± .359	.435 ± .010	.747 ± .249	.500 ± .293
	Low Intensity	.673 ± .188	.429 ± .150	.901 ± .249	.520 ± .292
Control	High Intensity	.596 ± .248	.520 ± .166	.663 ± .278	.609 ± .214
	Low Intensity	.530 ± .191	.543 ± .279	.637 ± .341	.571 ± .211

* Values $\overline{X}$ ± SD

intensity exercise group had lower post exercise levels of FFA after training ($p < 0.05$). The high intensity exercise group had lower post exercise levels of FFA after training although the change was not significant ($p > 0.05$) (Appendix C-6, C-7).

A three-way analysis of variance was used to determine the effect of training on resting and post exercise glucose values. The results are summarized in Table 7.

The high intensity groups had lower pre training resting glucose levels ($p < 0.05$) than the low intensity groups (Figure 10) indicating a "B" main effect. Resting glucose values (Figure 10) were lower after training for both the high and low intensity exercise and control groups ($p < 0.05$) indicating a "C" main effect (Appendix C-8, C-9).

The high intensity exercise group had lower post exercise glucose values ($p < 0.05$) than the high intensity control group both pre and post training (Figure 11). High intensity and low intensity control groups showed decreased post exercise glucose values ($p < 0.05$) from pre to post training (Figure 11). The low intensity groups showed a decrease ($p < 0.05$) in post exercise glucose values following training (Appendix C-10, C-11).

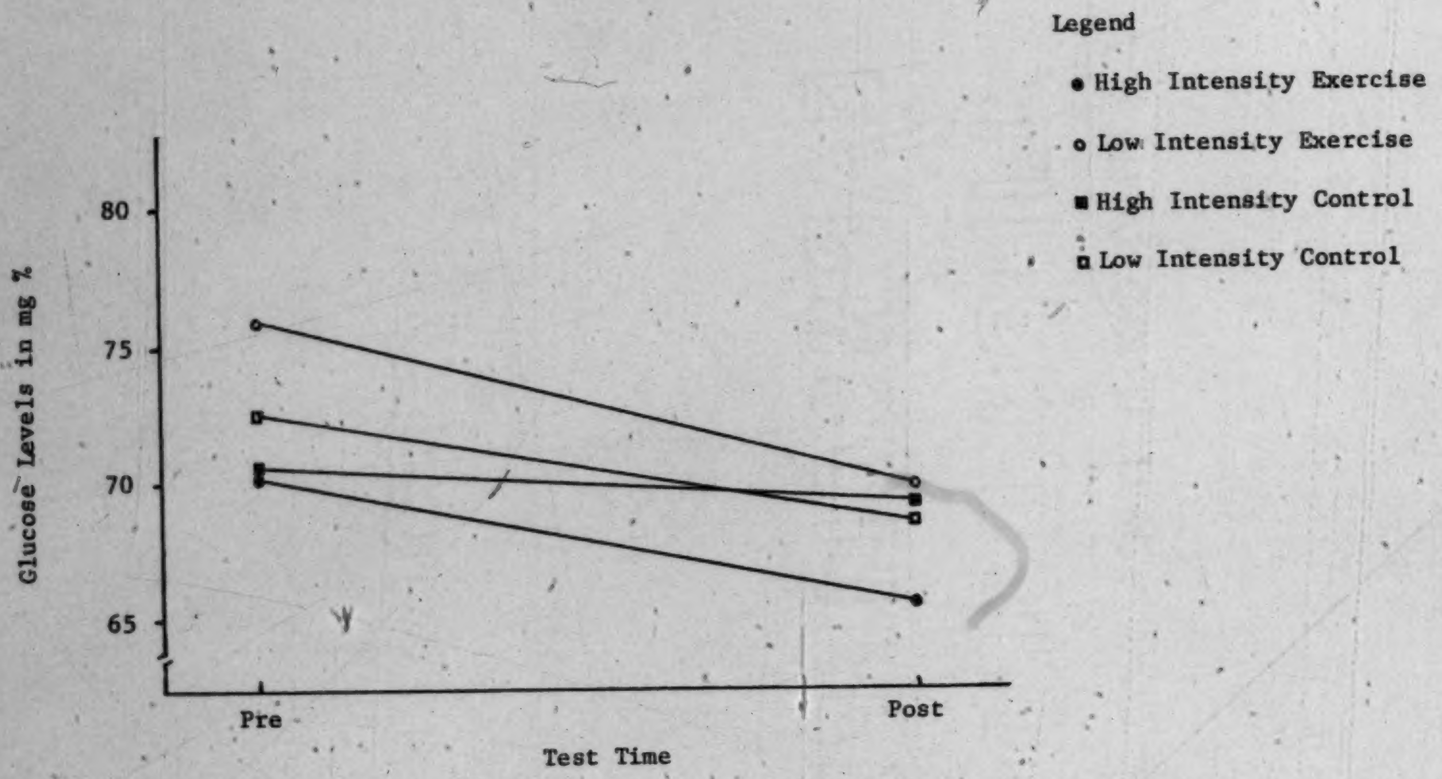

FIGURE 10, THE EFFECTS OF TRAINING ON RESTING BLOOD GLUCOSE LEVELS

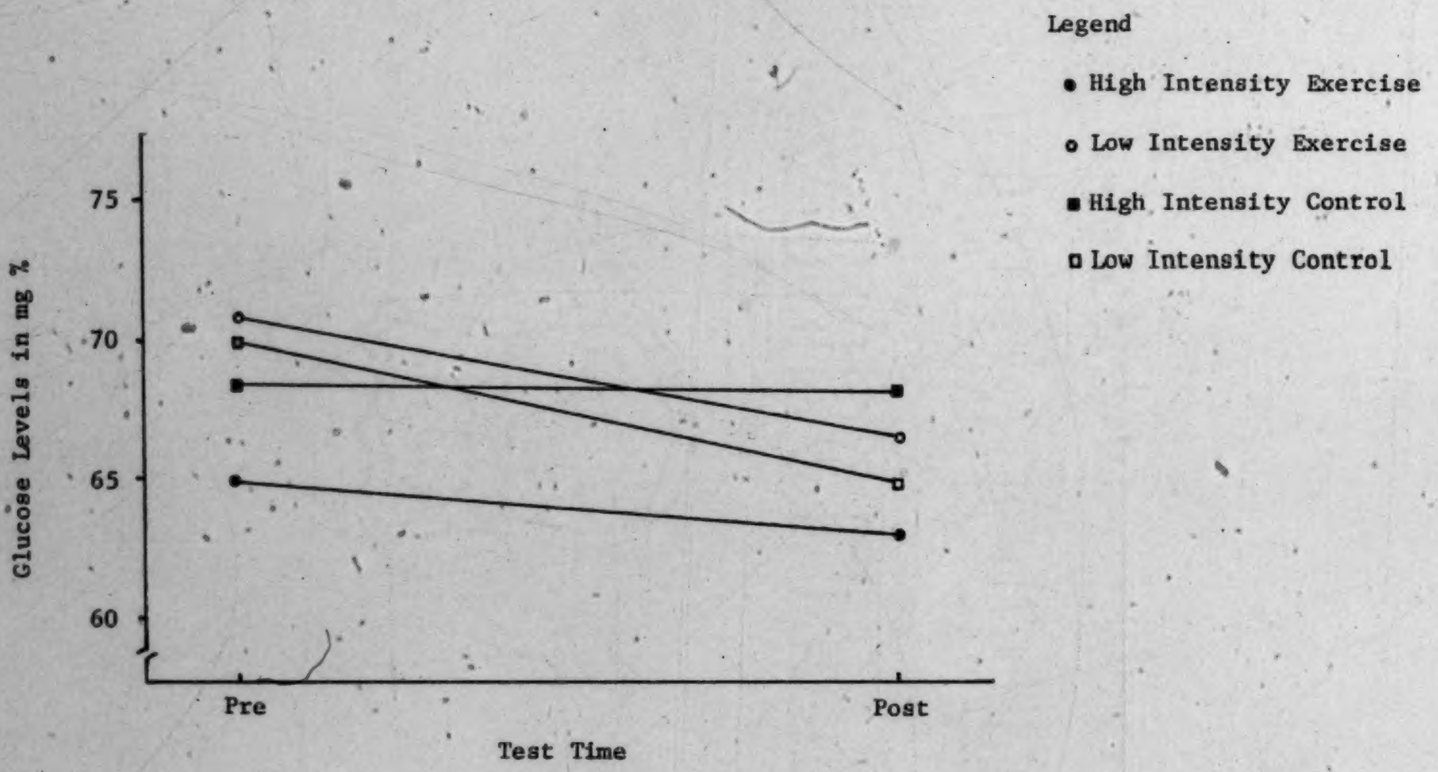

FIGURE 11. THE EFFECTS OF TRAINING ON POST EXERCISE BLOOD GLUCOSE LEVELS

TABLE 7

THE EFFECT OF TRAINING ON RESTING AND POST EXERCISE MEAN GLUCOSE LEVELS IN mg %*

		Resting		Post Exercise	
		Pre Training	Post Training	Pre Training	Post Training
Exercise	High Intensity	70.33 ± 5.06	65.78 ± 5.31	64.88 ± 4.50	62.66 ± 9.92
	Low Intensity	76.03 ± 6.29	69.39 ± 5.40	70.16 ± 4.23	66.42 ± 5.04
Control	High Intensity	70.30 ± 8.49	68.06 ± 5.53	71.39 ± 7.86	67.81 ± 6.20
	Low Intensity	73.72 ± 6.78	67.39 ± 4.21	69.84 ± 8.75	64.56 ± 7.49

* Values $\bar{X} \pm SD$

CHAPTER V

DISCUSSION

Prior to training, percent body fat of the three groups ranged from 27.22% to 27.39% which fall within the range of 20.73% to 38.60% fat reported in other research on sedentary young females (Young et al., 1961; Katch and Michael, 1968; Wilmore and Behnke, 1970; Johnson et al., 1972; Moody et al., 1972; Pollock et al., 1975; Smith, 1975; Wallace, 1975; Girandola, 1976; Smith, 1976; Shire, 1977; Novak, 1978; White, 1978). Obesity in women has been defined as greater than 30% body fat (Franklin et al., 1979). Thus, the women in this study were not obese but they were overweight.

After training, both the high and low intensity exercise groups showed significant decreases of 1.95% fat and 1.93% fat respectively. Similar decreases in percent body fat after training have been reported by Smith (1975), Johnson (1972), Girandola (1976), White (1978), and Massicotte et al. (1979).

There was no significant difference between high intensity and low intensity exercise groups in the decrease of percent body fat. Very few studies have examined the effect of intensity of exercise on body composition changes of women and conflicting results have been reported. Girandola (1976) equated high and low intensity exercise groups on total power output. The high intensity group showed a small but insignificant gain in percent body fat while the low intensity group showed a decrease ($p < 0.05$) of 1.1% in body fat. The high intensity group interval trained at a high intensity for a total of five minutes. Because it was anaerobic work of a short duration, it appeared to have little effect on energy expenditure. Smith (1975) found that a high intensity exercise group showed a greater decrease in percent body fat than a low intensity exercise group although the difference was not significant. Body density was predicted from skinfold measures. The results of this study are in agreement with Smith (1975).

The decrease in percent body fat represented an average loss of 2.9 pounds of fat in the low intensity group ($p < 0.05$) and an average loss of 2.44 pounds of fat in the high intensity group ($p > 0.05$). The control group showed a nonsignificant gain of 1.71 pounds of fat. Other studies have shown similar decreases in body fat after training periods of approximately the same length (Shire et al., 1977; Moody et al., 1972; Smith et al., 1975). The period of training promoted a decrease in total body fat of the exercise groups and prevented a fat gain as seen in the controls. Although the changes in body fat of both the high and low intensity exercise groups are similar, only the low intensity group showed a statistically significant decrease after training ("B" main effect). There was no significant difference between high and low intensity groups on post training body fat which would indicate that the two intensities of exercise had a similar effect on adipose depots.

The high and low intensity exercise groups experienced significant gains of 2.78 and 3.37 pounds of fat free weight after training while the control showed no change. There was no statistical difference between the groups in fat free weight gain indicating that the two intensities affected muscle hypertrophy in a similar manner. Most studies on women have shown slight but nonsignificant gains in lean body mass (Smith, 1975; Girandola, 1976; Zuti, 1976; Shire, 1977). Moody et al. (1972) found a significant increase in the fat free weight of obese subjects but not in normal weight subjects involved in the same program. Smith (1976) found fat free weight of sedentary subjects to increase 2.42 pounds ($p < 0.01$) after endurance training; however, body density was predicted from skinfold measures. A possible explanation for the increase of fat free weight in the present study is that the women worked against increasing workload using the large muscle mass of the legs. The overload principle was incorporated into the training program - as training heart rate adapted to a workload, the workload was increased to maintain the same relative intensity. Wilmore (1974) found lean body weight of women to increase ($p < 0.05$) 2.33 pounds after a ten week weight training program which resulted in significant gains in leg strength. In the present study, work against increasing resistances may have resulted in increased muscle hypertrophy of the leg muscles. The increases in fat free weight are greater than those reported on women but compare with fat free weight gains of men (Misner et al., 1974).

No significant changes in body weight occurred in any of the groups. A significant "A" main effect was obtained and indicated that there was a difference between groups on body weight. However a Scheffé post hoc comparison failed to show where the difference occurred. This may have occurred because the "A" main effect just reached significance and because the Scheffé method was used as it is more rigorous than other multiple comparison methods with regard to Type 1 errors (Ferguson, 1976). By examining the data (Table 4), it would appear that the control group was heavier than either exercise group both pre and post training.

The shift of body compartments masked a change in body weight as fat free weight has a greater density than fat. A decrease in body fat was offset by a concomitant increase in fat free weight. Studies of similar durations have also shown no change in body weight although body fat and fat free weight changed (Moody et al., 1972; Cunningham and Hill, 1975; Wallace, 1975; Smith, 1975; Girandola, 1976, White, 1978). Thus, in exercise weight control programs it is essential to measure body composition by an accurate method such as hydrostatic weighing so that changes in body fat and fat free weight can be seen. The purpose of weight reduction is to reduce body fat and not necessarily body weight.

$\dot{V}O_2$ max of untrained sedentary females, twenty to thirty years of age range from 29.5 ml/kg/min to 41.5 ml/kg/min (Kilbom, 1971; Edwards, 1974; Flint et al., 1974; Kearney et al., 1976; Rosentswieg, 1977; Lesmes et al., 1978; Novak et al., 1978; Pedersen and Jorgensen, 1978; Rosentswieg and Burrhus, 1978; Massicotte et al., 1979). $\dot{V}O_2$ max of the groups in this study ranged from 36.56 to 36.75 ml/kg/min and fell within the upper part of the reported range. Although the subjects were sedentary, they had moderate fitness levels.

Both high and low intensity exercise groups showed a slight but nonsignificant increase in $\dot{V}O_2$ max. Edwards (1974) trained sedentary females at a high intensity which corresponded to a heart rate of 145 beats per minute and a low intensity which corresponded to a heart rate of 125 beats per minute. Both groups showed significant increases in $\dot{V}O_2$ max following training. The heart rates used were approximately the same as were used in the present study (Appendix E); however the subjects had initially very low fitness levels reflected in an average $\dot{V}O_2$ max of 26.18 ml/kg/min. Because the subjects involved in this study had relatively high $\dot{V}O_2$ max for sedentary subjects, higher intensities, longer duration of training sessions or more frequent training sessions may have been needed to produce significant changes in $\dot{V}O_2$ max. Rosentswieg (1977) trained sedentary females for fifteen minutes three times per week at a heart rate of 160 beats/min and found a slight but nonsignificant increase in $\dot{V}O_2$ max after two months of training. By increasing the length of training to five times per week, significant changes in $\dot{V}O_2$ max occurred after two months of training. In the present study, the low intensity group trained at an average heart rate of 125.6 beats per minute (65% of maximum heart rate). It would appear that the intensity was not great enough to result in changes in $\dot{V}O_2$ max. The high intensity group trained at an average heart rate of 155.2 beats per minute (80% of maximum heart rate) for approximately fifteen to twenty minutes three times per week. Possibly the duration of training sessions was not long enough or frequent enough to cause significant changes in $\dot{V}O_2$ max for subjects of moderate fitness levels.

There was no significant difference between groups on resting lactate levels pre and post training. Values ranged from 19.91 to 27.22 mg % which are higher than the range of resting levels of 3.63 to 12.71 mg % reported in other studies (Boyd et al., 1974; Maughan et al., 1978; Foster, 1978). One possible explanation for the high lactate values is that the subjects rushed up stairs to the laboratory to make early morning appointments on time.

The high intensity exercise groups produced more lactate after exercise than the low intensity exercise groups both pre and post training (Figure 7). The high lactate values indicate that the high intensity exercise resulted in a greater recruitment of FG fibers and increased energy production by anaerobic glycolysis (Fox and Mathews, 1976). Pre training low intensity lactate levels ranged from 32.49 to 34.39 mg % while high intensity post exercise mean lactate concentrations ranged from 37.76 to 45.47 mg %. The values are similar to those of Kilbom (1971) measured on sedentary women at similar low and high intensities. Body et al. (1974) found that lactate levels of 54.48 to 81.72 mg % inhibited FFA mobilization. It would appear that high intensity exercise did not result in high enough lactate levels to inhibit FFA mobilization.

There was no significant difference between exercise and control groups for post exercise lactate values pre training. Following training, the exercise groups showed an adaptation to submaximal exercise as they produced significantly less lactate than the control groups ($p < 0.05$). The high intensity control group produced significantly more lactate than the high intensity exercise group. The lactate levels of the high intensity control group reached 55.74 mg % which may have been high enough to inhibit FFA mobilization.

Initial post exercise lactates were measured after the completion of a total power output of 12,000 kpm whereas post training lactates were measured after the completion of a total power output of 14,000 kpm. Following training, there was a slight but nonsignificant decrease in post exercise lactate levels in both exercise groups. An increased total power output was needed to cause the same increase in lactate production and reflected a training adaptation of muscle to submaximal exercise (Saltin and Karlsson, 1971).

There was no difference between the exercise and control groups on pre training resting FFA levels which ranged from 0.530 to 0.698 mM/1 and fell within the range found by Dole (1956), Gordon and Cherkes (1956) and Foster (1978). Post training resting FFA levels decreased significantly in both the high and low intensity exercise groups while the FFA levels of the control groups remained the same. Several researchers have reported similar decreases in resting FFA levels of trained subjects (Rennie et al., 1974; Bloom et al., 1976; Holloszy, 1977; Holloszy, 1978; Bransford, 1979).

Prior to training, both exercise groups had higher FFA concentrations after exercise than the control groups. There was no difference between the high and low intensity groups on FFA concentrations which would indicate that both intensities had a similar effect on FFA mobilization and uptake.

Following training, exercise resulted in elevated FFA levels in all the groups; however, both the exercise groups had significantly lower post exercise FFA concentrations than the control groups. Other studies have found trained individuals to have lower FFA concentrations during exercise than untrained subjects (Rennie et al., 1974; McCafferty and Horvath, 1977; Holloszy, 1977; Holloszy, 1978; Oscai, 1978; White et al., 1978; Bransford, 1979). Changes in FFA concentrations indicate an imbalance between rate of release of FFA from adipose tissue and rate of removal by working muscles (Issekutz et al., 1966). Some researchers have found that trained subjects have lower RER than untrained subjects while working at the same relative intensity which suggests an increased oxidative use of FFA (Christensen and Hansen, 1939; Holloszy, 1977; Saltin, 1977; Bransford, 1979).

Post exercise FFA concentrations of the low intensity exercise group decreased significantly from .901 mM/1 to .520 mM/1 following training while FFA levels of the high intensity group decreased from .747 mM/1 to .500 mM/1 ($p > 0.05$). The low intensity exercise group produced a greater change than the high intensity exercise group from pre to post training levels due to a higher pre training value. The FFA concentrations of both groups post training, post exercise are the same and indicate that the two intensities affected the mobilization and uptake of FFA by the cell in a similar manner. This may imply that there was increased oxidation of FFA in the trained individual. Further research is needed to measure FFA turnover rate to determine uptake and utilization of FFA in the cell.

Mean resting glucose levels ranged from 65.78 to 76.03 mg % and are in agreement with studies which determined glucose levels on whole blood (Wahren et al., 1971; Johnson and Rennie, 1973; Ahlborg et al., 1974; Foster, 1978; Maehlum et al., 1978). Average values for resting blood glucose levels determined on blood plasma are 88 to 90 mg % (Foster, 1978; Gollnick, 1978).

Pre training resting glucose levels of the high intensity groups were statistically lower than those of the low intensity groups. However, when the values of 70.32 mg % as compared to 74.87 mg % are considered in physiological terms, the difference is not significant. A difference of at least 10 mg % is needed in order for it to be considered significant physiologically (Udassin et al., 1977).

Resting glucose values were lower after training for both the high and low intensity exercise and control groups. Because there was no control of diet and the decrease occurred in both trained and untrained subjects, it is likely that the type or quantity of carbohydrate intake varied from pre to post tests and resulted in a slight but significant decrease in resting blood glucose (Thompson et al., 1978). Gollnick (1978), Bloom et al. (1976), Rennie et al. (1974), Sutton (1978), and Massicotte (1979) have found resting blood glucose levels of trained and untrained individuals to be the same.

Both high and low work intensities resulted in slight but nonsignificant decreases in blood glucose levels following exercise both pre and post training with the exception of the high intensity control group which showed an increase in pre training, post exercise values. The response of the low intensity groups agree with previous research which has shown no significant change in blood glucose levels of untrained individuals during moderate intensity exercise (Wahren, 1971; Rottini et al., 1971; Johnson andRennie, 1973; Udassin, 1977; White et al., 1978; Sutton, 1978). During high intensity work, most studies have shown blood glucose levels to rise with the increase being greater in trained than untrained subjects (Pruett, 1970; Wahren, 1971; Bloom et al., 1976; White et al., 1978; Sutton, 1978). In the present study, the high intensity exercise group had lower post exercise glucose values than the high intensity control group both pre and post training. The high intensity control group showed a slight increase in post exercise pre training glucose levels. These results contradict the findings reported in the literature. However high intensity exercise at 80% of maximum heart rate may not have been great enough to result in increased glucose levels. Wahren (1971) found that power outputs of 800 to 1200 kpm/min resulted in increased glucose levels with the greatest increase at the highest power output. Typical power outputs for the high intensity group ranged from 450 to 900 kpm/min (Appendix G). Very few of the subjects could maintain the higher power outputs for extended periods of time.

By examining the results of FFA, it can be seen that there was little difference between high and low intensity exercise groups on the metabolic response to exercise. The same could apply to the effect of exercise on glucose and thus, would explain why glucose levels of the high intensity groups did not increase.

Post exercise glucose levels were measured three minutes after the cessation of exercise. Insulin levels have been found to increase rapidly within two to ten minutes after exercise is terminated (Pruett, 1971; Felig and Wahren, 1975). High rebound insulin levels could have contributed to decreased glucose levels.

The measurement of glucose, FFA and lactate help to explain why there was no significant difference between high and low intensity exercise groups on changes in body fat. Although lactate levels were higher in the high intensity exercise groups, they were not high enough to inhibit FFA mobilization. Resting and post exercise FFA concentrations decreased to similar levels in both the high and low intensity exercise groups indicating that the two intensities resulted in similar adaptations in FFA mobilization and uptake.

CHAPTER VI

SUMMARY AND CONCLUSIONS

The purpose of the study was to determine the effects of training at high or low relative intensities on body composition changes of women. Another purpose was to examine the effect of training at high and low relative intensities on glucose, lactate and FFA concentrations at rest and immediately after exercise.

Thirty-four female subjects were ranked according to percent body fat and $\dot{V}O_2$ max and were randomly assigned to either a high intensity group which worked at 80% of maximum heart rate, a low intensity group which worked at 65% of maximum heart rate or a control group which maintained their usual exercise and eating habits. The training program consisted of three training sessions per week for nine weeks. Groups were equated on total power output of 12,000 kpm for four weeks after which it was increased to 14,000 kpm.

Changes in body composition were measured by hydrostatic weighing. Blood samples were taken pre and post exercise during the first and last training sessions. A two-way analysis of variance was used to analyse data on body composition and $\dot{V}O_2$ max. A three-way analysis of variance was used to determine differences in pre and post training glucose, lactate and FFA concentrations.

Conclusions

Within the limits of this study the following conclusions were drawn:

1. There was no difference in the decrease of percent body far or the

increase in fat free weight produced by training at relative intensities of 65% or 80% of maximum heart rate equated on total power output.

2. Both the low and high intensity exercise programs resulted in a reduction of body fat, and therefore, either intensity could be used as an effective part of weight reduction programs for women with similar percent body fat and fitness levels.
3. Changes in body composition did not alter body weight.
4. Training at relative intensities of 65% or 80% of maximum heart rate showed no statistically significant change in $\dot{V}O_2$ max.
5. There was no difference between the lowered resting FFA levels produced by training at relative intensities of 65% or 80% of maximum heart rate.
6. Training at relative intensities of 65% or 80% of maximum heart rate resulted in similar decreases in post exercise FFA levels.
7. An increased total power output was needed to attain the same rate of lactate production in the trained as compared to the untrained state.

Recommendations

1. The FFA assay is a very long and complicated process with many steps which have the potential to lead to errors. Five to six months is needed to become proficient at the assay. Researchers should be aware of this before using the assay as part of a study.
2. More research is needed to examine the effect of different intensities of exercise on body composition.

3. The probability level for metabolic adaptations should be set at a higher level than $p < 0.05$ in order to establish some physiological changes occurring.
4. Because diet is an important factor affecting body composition, diet should be monitored more closely.

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APPENDIX A

SCREENING QUESTIONNAIRE

SCREENING QUESTIONNAIRE

NAME: ______________________________

AGE: ____________ HEIGHT: ____________ WEIGHT: ____________

ADDRESS: ______________________________
____
____

PHONE ____________ HOURS TO AVOID ____________

1. Has your weight changed significantly in the past 2 months?
   a) Yes b) No If (a), how much weight did you gain or lose? ____________
   Any significant reason known for the change (eg., illness, dieting, etc.) ______________________________

2. Do you smoke:
   a) Yes ____________ b) No ____________

3. How often are you involved in strenuous physical activity?
   a) daily b) 3-4 times/week c) once a week
   d) 1 or 2 times per month e) less than once a month
   Type ______________________________
   Duration: ______________________________

4. Have you had a medical examination within the last year?
   a) Yes b) No

5. Do you take any medicines, drugs, vitamins, etc.?
   a) Yes b) No, not at all
   If (a) specify: 1. regularly
   2. within past 3 days
   3. occasionally
   Tranquilizers ______
   Sedatives or hypnotics ______
   Antibiotics ______
   Anti-convulsants other than dilantin ______
   Dilantin ______
   Anti-hypertensives ______

Anti-coagulants _____

Cardiac Medication _____
(eg. digitalis, xylocaine, atropine)

Appetite depressants _____

Amphetamine, dexedrine, or
other CNS stimulants _____

Diruetics _______

Cholesterol-depressants _____

Anti-diabetic agents _____

Thyroid _____

Anti-thyroid preparations _____

Oral contraceptives _____

Hormones _____

Vitamins _____ Type ______________________

Other medications, including injections:

____

____

Common sense is your best guide in answering these few questions. Please read them carefully and check ✓ Yes opposite the question if it applies to you.

Yes

6. Has your doctor ever said you have heart trouble?

7. Do you frequently have pains in your heart and chest?

8. Do you often feel faint or have spells of severe dizziness?

9. Has a doctor ever said your blood pressure was too high?

10. Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggrevated by exercise, or might be made worse with exercise?

11. Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?

APPENDIX B

APPENDIX B-1. SUMMARY OF TWO-WAY ANOV ON PERCENT BODY FAT

APPENDIX B-2. SCHEFFÉ COMPARISON FOR % BODY FAT

APPENDIX B-3. SUMMARY OF TWO-WAY ANOV ON POUNDS FAT

APPENDIX B-4. SCHEFFÉ COMPARISON FOR POUNDS FAT

APPENDIX B-5. SUMMARY OF TWO-WAY ANOV ON FAT FREE WEIGHT

APPENDIX B-6. SCHEFFÉ COMPARISON FOR FAT FREE WEIGHT

APPENDIX B-7. SUMMARY OF TWO-WAY ANOV ON WEIGHT IN POUNDS

APPENDIX B-8. SCHEFFÉ COMPARISON ON WEIGHT IN POUNDS

APPENDIX B-9. SUMMARY OF TWO-WAY ANOV ON $\dot{V}O_2$ MAX

APPENDIX B-10. SCHEFFÉ COMPARISON ON $\dot{V}O_2$ MAX

APPENDIX B-1

SUMMARY OF TWO-WAY ANOV ON PERCENT BODY FAT

SOURCE OF VARIATION	SS	DF	MS	F	P
Between Subjects	858.867	29			
'A' Main Effects	35.234	2	17.617	0.578	0.5680699
Subjects Within Groups	823.621	27	30.504		
Within Subjects	116.027	30			
'B' Main Effects	21.367	1	21.367	7.418	0.0111848*
'A*B' Interaction	16.875	2	8.438	2.929	0.0705901
'B' x Subjects Within Groups	77.777	27	2.881		

*Significant $p < 0.05$

APPENDIX B-2

SCHEFFE COMPARISON FOR PERCENT FAT

	A 27.39	B 27.23	C 27.80	D 25.43	E 25.29	F 28.11
A 27.39	0	0.29	0.29	6.73*	7.65*	0.90
B 27.23		0	0.56	5.62	6.71*	1.34
C 27.80			0	9.75*	10.93*	0.17
D 25.43				0	0.03	12.47*
E 25.29					0	13.80*
F 28.11						0

$F^1 = 6.70$
$*p < 0.05$

APPENDIX B-3

SUMMARY OF TWO-WAY ANOV ON POUNDS FAT

SOURCE OF VARIATION	SS	DF	MS	F	P
Between Subjects	3742.188	29			
'A' Main Effects	499.180	2	249.590	2.078	0.1447446
Subjects Within Groups	3243.000	27	120.111		
Within Subjects	230.875	30			
'B' Main Effects	21.836	1	21.836	4.078	0.0534652*
'A*B' Interaction	64.453	2	32.227	6.019	0.0068918*
'B' x Subjects Within Groups	144.563	27	5.354		

*Significant $p < 0.05$

APPENDIX B-4

SCHEFFÉ COMPARISON FOR POUNDS FAT

		A	B	C	D	E	F
		35.07	34.12	38.44	32.63	31.22	40.15
A	35.07	0	0.84	10.61*	5.55	13.85*	24.12*
B	34.12		0	17.44*	2.07	7.86*	33.98*
C	38.44			0	31.56*	47.38*	2.78
D	32.63				0	1.86	52.85*
E	31.22					0	74.53*
F	40.15						0

$F^1 = 6.70$
* $p < 0.05$

APPENDIX B-5

SUMMARY OF TWO-WAY ANOV ON FAT FREE WEIGHT

SOURCE OF VARIATION	SS	DF	MS	F	P
Between Subjects	4908.438	29			
'A' Main Effects	509.492	2	254.746	1.564	0.2277662
Subjects Within Groups	4399.063	27	162.928		
Within Subjects	225.063	30			
'B' Main Effects	69.883	1	69.883	14.598	0.0007106*
'A*B' Interaction	26.289	2	13.145	2.746	0.0821260
'B' x Subjects Within Groups	129.250	27	4.787		

*Significant $p < 0.05$

APPENDIX B-6

SCHEFFÉ COMPARISONS FOR FAT FREE WEIGHT

	A	B	C	D	E	F
	92.70	91.54	99.63	95.48	94.91	99.96
A 92.70	-	-	50.16*	8.07*	-	55.05*
B 91.54		-	68.37*	16.21*	11.86*	74.76*
C 99.63			-	17.99*	23.27*	-
D 95.48				-	-	20.96*
E 94.91					-	26.64*
F 99.96						-

1
F = 6.70
* $p < 0.05$

APPENDIX B-7

SUMMARY OF TWO-WAY ANOV ON WEIGHT IN POUNDS

SOURCE OF VARIATION	SS	DF	MS	F	P
Between Subjects	12178.688	29			
'A' Main Effects	2380.625	2	1190.313	3.281	0.05*
Subjects Within Groups	9796.500	27	362.833		
Within Subjects	121.563	30			
'B' Main Effects	5.000	1	5.000	1.169	0.2890769
"A*B' Interaction	1.250	2	0.625	0.146	0.8646815
'B' x Subjects Within Groups	115.438	27	4.275		

*Significant $p < 0.05$

APPENDIX B-8

SCHEFFÉ COMPARISON ON WEIGHT IN POUNDS

		A	B	C	D	E	F
		127.70	124.10	138.60	128.10	124.40	139.60
A	127.70	0.00	0.18	1.64	0.00	0.15	1.95
B	124.10		0.00	2.90	0.22	0.00	3.31
C	138.60			0.00	1.52	2.79	0.00
D	128.10				0.00	0.19	1.82
E	124.40					0.00	3.18
F	139.60						0.00

$F^1 = 6.70$
* $p < 0.05$

APPENDIX B-9

SUMMARY OF TWO-WAY ANOV ON $\dot{V}O_2$ MAX

SOURCE OF VARIATION	SS	DF	MS	F	P
Between Subjects	758.813	29			
'A' Main Effects	75.977	2	37.988	1.502	0.2406778
Subjects Within Groups	682.813	27	25.289		
Within Subjects	327.250	30			
'B' Main Effects	6.055	1	6.055	0.667	0.4211029
'A*B' Interaction	76.211	2	38.105	4.200	0.0258058*
'B' x Subjects Within Groups	244.938	27	9.072		

*Significant $p < 0.05$

APPENDIX B-10

SCHEFFÉ COMPARISON ON $\dot{V}O_2$ MAX

	A 36.42	B 36.75	C 36.56	D 39.08	E 38.51	F 34.05
A 36.42	0.00	0.06	0.01	3.89	2.41	3.09
B 36.75		0.00	0.02	2.99	1.71	4.02
C 36.56			0.00	3.50	2.10	3.47
D 39.08				0.00	0.18	13.95*
E 38.51					0.00	10.97*
F 34.05						0.00

$F^1 = 6.70$
* $p < 0.05$

APPENDIX C

APPENDIX C-1. SUMMARY OF THREE-WAY ANOV ON RESTING LACTATES PRE AND POST TRAINING

APPENDIX C-2. SUMMARY OF THREE-WAY ANOV ON POST EXERCISE LACTATES PRE AND POST TRAINING

APPENDIX C-3. POST HOC CONTRASTS ON POST EXERCISE LACTATES PRE AND POST TRAINING

APPENDIX C-4. SUMMARY OF THREE-WAY ANOV ON RESTING FFA LEVELS PRE AND POST TRAINING

APPENDIX C-5. POST HOC CONTRACTS ON RESTING FFA LEVELS PRE AND POST TRAINING

APPENDIX C-6. SUMMARY OF THREE-WAY ANOV ON POST EXERCISE FFA LEVELS PRE AND POST TRAINING

APPENDIX C-7. POST HOC CONTRASTS ON POST EXERCISE FFA LEVELS PRE AND POST TRAINING

APPENDIX C-8. SUMMARY OF THREE-WAY ANOV ON RESTING GLUCOSE LEVELS PRE AND POST TRAINING

APPENDIX C-9. POST HOC CONTRASTS ON RESTING GLUCOSE LEVELS PRE AND POST TRAINING

APPENDIX C-10. SUMMARY OF THREE-WAY ANOV ON POST EXERCISE GLUCOSE LEVELS PRE AND POST TRAINING

APPENDIX C-11. POST HOC CONTRASTS ON POST EXERCISE GLUCOSE LEVELS PRE AND POST TRAINING

APPENDIX C-1

SUMMARY OF THREE-WAY ANOV ON RESTING LACTATES PRE AND POST TRAINING

SOURCE	SS	DF	MS	F	P
Between Subjects	3381.426	39			
A	112.773	1	112.773	1.27	0.267
B	0.801	1	0.801	0.01	0.925
AB	79.910	1	79.910	0.90	0.348
Subjects Within Group	3187.941	36	88.554		
Within Subjects	2903.914	40			
C	227.125	1	227.125	3.90	0.056
AC	0.547E-01	1	0.547E-01	0.00	0.978
BC	27.160	1	27.160	0.38	0.540
ABC	39.660	1	39.660	0.56	0.460
C x Subjects Within Group	2559.972	36	71.110		

APPENDIX C-2

SUMMARY OF THREE-WAY ANOV ON POST EXERCISE LACTATES PRE AND POST TRAINING

SOURCE	SS	DF	MS	F	P
Between Subjects	9063.062	39			
A	1496.562	1	1496.562	10.48	0.003*
B	1981.750	1	1981.750	13.87	0.001*
AB	442.500	1	442.500	3.10	0.087
Subjects Within Group	5142.250	36	142.840		
Within Subjects	4844.500	40			
C	159.875	1	159.875	1.35	0.252
AC	297.500	1	297.500	2.52	0.121
BC	64.563	1	64.563	0.55	0.465
ABC	67.813	1	67.813	0.57	0.454
C x Subjects Within Group	4254.650	36	118.188		

*$p < 0.05$

APPENDIX C-3

POST HOC CONTRASTS ON POST EXERCISE LACTATES PRE AND POST TRAINING

Post-hoc contrasts among levels of A for given levels of B: Table AB

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-High) 1 1		(Control-High) 2 1	-13.35	142.84	1.	36.	12.485	0.001*
(Exer-Low) 1 2		(Control-Low) 2 2	-3.95	142.84	1.	36.	1.090	0.303

Post-hoc contrasts among levels of B for given levels of A: Table AB

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-High) 1 1		(Exer-Low) 1 2	5.25	142.84	1.	36.	1.930	0.173
(Control-High) 2 1		(Control-Low) 2 2	14.66	142.84	1.	36.	15.049	0.000*

Post-hoc contrasts among levels of A for given levels of C: Table AC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-Pre) 1 1		(Control-Pre) 2 1	-4.79	130.51	1.	72.	1.760	0.189
(Exer-Post) 1 2		(Control-Post) 2 2	-12.51	130.51	1.	72.	11.986	0.001*

Post-hoc contrasts among levels of C for given levels of A: Table AC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-Pre) 1 1		(Exer-Post) 1 2	1.03	118.19	1.	36.	0.090	0.766
(Control-Pre) 2 1		(Control-Post) 2 2	-6.69	118.19	1.	36.	3.782	0.060

Post-hoc contrasts among levels of b for given levels of C: Table BC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(High-Pre) 1 1		(Low-Pre) 2 1	8.16	130.51	1.	72.	5.099	0.027*
(High-Post) 1 2		(Low-Post) 2 2	11.75	130.51	1.	72.	10.579	0.002*

Post-hoc contrasts among levels of C for given levels of B: Table BC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(High-Pre) 1 1		(High-Post) 1 2	-4.62	118.19	1.	36.	1.810	0.187
(Low-Pre) 2 1		(Low-Post) 2 2	-1.03	118.19	1.	36.	0.090	0.766

*$p < 0.05$

APPENDIX C-3 (Continued)

CELL MEANS TABLE AB

Column	1	2	3
	Exer-High	Exer-Low	
Row 1	37.222	31.972	34.597
	Control-High	Control-Low	
Row 2	50.577	35.918	43.248
Row 3	43.900	33.945	38.923

CELL MEANS TABLE AC

Column	1	2	3
	Exer-Pre	Exer-Post	
Row 1	35.112	34.083	34.597
	Control-Pre	Control-Post	
Row 2	39.905	46.590	43.248
Row 3	37.905	46.590	43.248

CELL MEANS TABLE BC

Column	1	2	3
	High-Pre	High-Post	
Row 1	41.587	46.212	43.900
	Low-Pre	Low-Post	
Row 2	33.429	34.461	33.945
Row 3	37.508	40.337	38.923

APPENDIX C-4

SUMMARY OF THREE-WAY ANOV ON RESTING FFA LEVELS PRE AND POST TRAINING

SOURCE	SS	DF	MS	F	P
Between Subjects	1.487	39			
A	0.153E-03	1	0.153E-03	0.00	0.952
B	0.351E-03	1	0.351E-03	0.01	0.927
AB	0.565E-03	1	0.565E-03	0.01	0.908
Subjects Within Group	1.486	36	0.413E-01		
Within Subjects	2.607	40			
C	0.329	1	0.329	6.08	0.019*
AC	0.284	1	0.284	5.25	0.028*
BC	0.249E-01	1	0.249E-01	0.46	0.502
ABC	0.202E-01	1	0.202E-01	0.37	0.545
C x Subjects Within Group	1.949	36	0.541E-01		

*$p < 0.05$

APPENDIX C-5

POST HOC CONTRASTS ON RESTING FFA LEVELS PRE AND POST TRAINING

Post-hoc contrasts among levels of A for given levels of B: Table AB

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-High) 1 1		(Control-High) 2 1	0.00	0.04	1.	36.	0.002	0.969
(Exer-Low) 1 2		(Control-Low) 2 2	-0.01	0.04	1.	36.	0.016	0.902

Post-hoc contrasts among levels of B for given levels of A: Table AB

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-High) 1 1		(Exer-Low) 1 2	0.01	0.04	1.	36.	0.022	0.883
(Control-High) 2 1		(Control-Low) 2 2	-0.00	0.04	1.	36.	0.000	0.988

Post-hoc contrasts among levels of A for given levels of C: Table AC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-Pre) 1 1		(Control-Pre) 2 1	0.12	0.05	1.	72.	2.845	0.096
(Exer-Post) 1 2		(Control-Post) 2 2	-0.12	0.05	1.	72.	3.120	0.082

Post-hoc contrasts among levels of C for given levels of A: Table AC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-Pre) 1 1		(Exer-Post) 1 2	0.25	0.05	1.	36.	11.316	0.002*
(Control-Pre) 2 1		(Control-Post) 2 2	0.01	0.05	1.	36.	0.015	0.903*

Post-hoc contrasts among levels of B for given levels of C: Table BC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(High-Pre) 1 1		(Low-Pre) 2 1	0.04	0.05	1.	72.	0.327	0.369
(High-Post) 1 2		(Low-Post) 2 2	-0.03	0.05	1.	72.	0.201	0.655

Post-hoc contrasts among levels of C for given levels of B: Table BC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(High-Pre) 1 1		(High-Post) 1 2	0.16	0.05	1.	36.	4,938	0.033*
(Low-Pre) 2 1		(Low-Post) 2 2	0.09	0.05	1.	36.	1.598	0.214

$^{*}p < 0.05$

APPENDIX C-5 (Continued)

CELL MEANS TABLE AB

Column	1	2	3
	Exer-High	Exer-Low	
Row 1	0.560	0.551	0.556
	Control-High	Control-Low	
Row 2	0.558	0.559	0.558
Row 3	0.559	0.555	0.557

CELL MEANS TABLE AC

Column	1	2	3
	Exer-Pre	Exer-Post	
Row 1	0.679	0.432	0.556
	Control-Pre	Control-Post	
Row 2	0.563	0.554	0.558
Row 3	0.621	0.493	0.557

CELL MEANS TABLE BC

Column	1	2	3
	High-Pre	High-Post	
Row 1	0.641	0.477	0.559
	Low-Pre	Low-Post	
Row 2	0.601	0.508	0.555
Row 3	0.621	0.493	0.557

APPENDIX C-6

SUMMARY OF THREE-WAY ANOV ON POST EXERCISE FFA LEVELS PRE AND POST TRAINING

SOURCE	SS	DF	MS	F	P
Between Subjects	2.633	39			
A	0.442E-01	1	0.442E-01	0.64	0.431
B	0.151	1	0.151	0.22	0.644
AB	0.708E-01	1	0.708E-01	1.02	0.320
Subjects Within Group	2.503	36	0.695E-01		
Within Subjects	3.775	40			
C	0.699	1	0.699	9.30	0.004*
AC	0.323	1	0.323	4.29	0.046*
BC	0.267E-01	1	0.267E-01	0.35	0.555
ABC	0.185E-01	1	0.185E-01	0.25	0.622
C x Subjects Within Group	2.708	36	0.752E-01		

*$p < 0.05$

APPENDIX C-7

POST HOC CONTRASTS ON POST EXERCISE FFA LEVELS PRE AND POST TRAINING

Post-hoc contrasts among levels of A for given levels of B: Table AB

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-High) 1 1		(Control-High) 2 1	-0.01	0.07	1.	36.	0.022	0.882
(Exer-Low) 1 2		(Control-Low) 2 2	0.11	0.07	1.	36.	1.631	0.210

Post-hoc contrasts among levels of B for given levels of A: Table AB

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-High) 1 1		(Exer-Low) 1 2	-0.09	0.07	1.	36.	1.089	0.304
(Control-High) 2 1		(Control-Low) 2 2	0.03	0.07	1.	36.	0.147	0.703

Post-hoc contrasts among levels of A for given levels of C: Table AC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-Pre) 1 1		(Control-Pre) 2 1	0.17	0.07	1.	72.	4.183	0.044*
(Exer-Post) 1 2		(Control-Post) 2 2	-0.08	0.07	1.	72.	0.884	0.350

Post-hoc contrasts among levels of C for given levels of A: Table AC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-Pre) 1 1		(Exer-Post) 1 2	0.31	0.08	1.	36.	13.109	0.001*
(Control-Pre) 2 1		(Control-Post) 2 2	0.06	0.08	1.	36.	0.479	0.493

Post-hoc contrasts among levels of B for given levels of C: Table BC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(High-Pre) 1 1		(Low-Pre) 2 1	-0.06	0.07	1.	72.	0.566	0.454
(High-Post) 1 2		(Low-Post) 2 2	0.01	0.07	1.	72.	0.011	0.916

Post-hoc contrasts among levels of C for given levels of B: Table BC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(High-Pre) 1 1		(High-Post) 1 2	0.15	0.08	1.	36.	3.012	0.091
(Low-Pre) 2 1		(Low-Post) 2 2	0.22	0.08	1.	36.	6.642	0.014*

$^*p < 0.05$

APPENDIX C-7 (Continued).

CELL MEANS TABLE AB

Column	1	2	3
	Exer-High	Exer-Low	
Row 1	0.623	0.710	0.667
	Control-High	Control-Low	
Row 2	0.636	0.604	0.620
Row 3	0.630	0.657	0.643

CELL MEANS TABLE AC

Column	1	2	3
	Exer-Pre	Exer-Post	
Row 1	0.824	0.510	0.667
	Control-Pre	Control-Post	
Row 2	0.650	0.590	0.620
Row 3	0.737	0.550	0.643

CELL MEANS TABLE BC

Column	1	2	3
	High-Pre	High-Post	
Row 1	0.641	0.477	0.559
	Low-Pre	Low-Post	
Row 2	0.769	0.545	0.657
Row 3	0.737	0.550	0.643

APPENDIX C-8

SUMMARY OF THREE-WAY ANOV ON RESTING GLUCOSE LEVELS PRE AND POST TRAINING

SOURCE	SS	DF	MS	F	P
Between Subjects	1652.350	39			
A	4.312	1	4.312	0.11	0.743
B	173.250	1	173.250	4.40	0.043*
AB	58.000	1	58.000	1.47	0.233
Subjects Within Group	1416.812	36	39.356		
Within Subjects	1662.625	40			
C	504.188	1	504.188	16.42	0.000*
AC	6.250	1	6.250	0.20	0.655
BC	42.563	1	42.563	1.39	0.247
ABC	4.125	1	4.125	0.13	0.716
C x Subjects Within Group	1105.500	36	30.708		

*$p < 0.05$

APPENDIX C-9

POST HOC CONTRASTS ON RESTING GLUCOSE LEVELS PRE AND POST TRAINING

Post-hoc contrasts among levels of A for given levels of B: Table AB

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-High) 1 1		(Control-High) 2 1	-1.27	39.36	1.	36.	0.411	0.525
(Exer-Low) 1 2		(Control-Low) 2 2	2.15	39.36	1.	36.	1.175	0.286

Post-hoc contrasts among levels of B for given levels of A: Table AB

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-High) 1 1		(Exer-Low) 1 2	-4.65	39.36	1.	36.	5.495	0.025*
(Control-High) 2 1		(Control-Low) 2 2	-1.23	39.36	1.	36.	0.383	0.540

Post-hoc contrasts among levels of A for given levels of C: Table AC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-Pre) 1 1		(Control-Pre) 2 1	1.02	35.03	1.	72.	0.297	0.587
(Exer-Post) 1 2		(Control-Post) 2 2	-0.14	35.03	1.	72.	0.006	0.940

Post-hoc contrasts among levels of C for given levels of A: Table AC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-Pre) 1 1		(Exer-Post) 1 2	5.60	30.71	1.	36.	10.210	0.003*
(Control-Pre) 2 1		(Control-Post) 2 2	4.44	30.71	1.	36.	6.414	0.016*

Post-hoc contrasts among levels of B for given levels of C: Table BC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(High-Pre) 1 1		(Low-Pre) 2 1	-4.41	35.03	1.	72.	5.544	0.021*
(High-Post) 1 2		(Low-Post) 2 2	-1.47	35.03	1.	72.	0.618	0.434

Post-hoc contrasts among levels of C for given levels of B: Table BC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(High-Pre) 1 1		(High-Post) 1 2	3.55	30.71	1.	36.	4.106	0.050*
(Low-Pre) 2 1		(Low-Post) 2 2	6.49	30.71	1.	36.	13.701	0.001*

* $p < 0.05$

APPENDIX C-9 (Continued)

CELL MEANS TABLE AB

Column	1	2	3
	Exer-High	Exer-Low	
Row 1	68.057	72.707	70.382
	Control-High	Control-Low	
Row 2	69.329	70.557	69.943
Row 3	68.693	71.632	70.162

CELL MEANS TABLE AC

Column	1	2	3
	Exer-Pre	Exer-Post	
Row 1	73.182	67.582	70.382
	Control-Pre	Control-Post	
Row 2	72.162	67.724	69.943
Row 3	72.672	67.653	70.162

CELL MEANS TABLE BC

Column	1	2	3
	High-Pre	High-Post	
Row 1	70.468	66.197	68.693
	Low-Pre	Low-Post	
Row 2	74.875	68.389	71.632
Row 3	72.672	67.653	70.162

APPENDIX C-10

SUMMARY OF THREE-WAY ANOV ON POST EXERCISE GLUCOSE LEVELS PRE AND POST TRAINING

SOURCE	SS	DF	MS	F	P
Between Subjects	2577.188	39			
A	112.500	1	112.500	1.84	0.184
B	22.750	1	22.750	0.37	0.546
AB	238.938	1	238.938	3.90	0.056
Subjects Within Group	2203.000	36	61.194		
Within Subjects	1646.812	40			
C	273.875	1	273.875	7.30	0.010*
AC	10.188	1	10.188	0.27	0.605
BC	12.563	1	12.563	0.34	0.566
ABC	0.375	1	0.375	0.01	0.921
C x Subjects Within Group	1349.813	36	37.495		

*$p < 0.05$

APPENDIX C-11

POST HOC CONTRASTS ON POST EXERCISE GLUCOSE LEVELS PRE AND POST TRAINING

Post-hoc contrasts among levels of A for given levels of B: Table AB

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-High) 1 1		(Control-High) 2 1	-5.83	61.19	1.	36.	5.550	0.024*
(Exer-Low) 1 2		(Control-Low) 2 2	1.09	61.19	1.	36.	0.194	0.662

Post-hoc contrasts among levels of B for given levels of A: Table AB

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-High) 1 1		(Exer-Low) 1 2	-4.52	61.19	1.	36.	3.337	0.076
(Control-High) 2 1		(Control-Low) 2 2	2.40	61.19	1.	36.	0.940	0.339

Post-hoc contrasts among levels of A for given levels of C: Table AC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-Pre) 1 1		(Control-Pre) 2 1	-3.09	49.34	1.	72.	1.939	0.168
(Exer-Post) 1 2		(Control-Post) 2 2	-1.65	49.34	1.	72.	0.549	0.461

Post-hoc contrasts among levels of C for given levels of A: Table AC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(Exer-Pre) 1 1		(Exer-Post) 1 2	2.97	37.49	1.	36.	2.360	0.133
(Control-Pre) 2 1		(Control-Post) 2 2	4.42	37.49	1.	36.	5.216	0.028*

Post-hoc contrasts among levels of B for given levels of C: Table BC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(High-Pre) 1 1		(Low-Pre) 2 1	-1.86	49.34	1.	72.	0.703	0.404
(High-Post) 1 2		(Low-Post) 2 2	-0.26	49.34	1.	72.	0.013	0.908

Post-hoc contrasts among levels of C for given levels of B: Table BC

CELL	VS	CELL	MEAN DIFF	MEAN SQ.	DF1	DF2	F	P
(High-Pre) 1 1		(High-Post) 1 2	2.90	37.49	1.	36.	2.237	0.143
(Low-Pre) 2 1		(Low-Post) 2 2	4.50	37.49	1.	36.	5.404	0.026*

*$p < 0.05$

APPENDIX C-11 (Continued)

CELL MEANS TABLE AB

Column	1	2	3
	Exer-High	Exer-Low	
Row 1	63.770	68.289	66.030
	Control-High	Control-Low	
Row 2	69.598	67.200	68.399
Row 3	66.684	67.745	67.214

CELL MEANS TABLE AC

Column	1	2	3
	Exer-Pre	Exer-Post	
Row 1	67.517	64.542	66.030
	Control-Pre	Control-Post	
Row 2	70.610	66.188	68.399
Row 3	69.064	65.365	67.214

CELL MEANS TABLE BC

Column	1	2	3
	High-Pre	High-Post	
Row 1	68.132	65.236	66.684
	Low-Pre	Low-Post	
Row 2	69.995	65.494	67.745
Row 3	69.064	65.365	67.214

APPENDIX D

APPENDIX D-1. RAW DATA FOR THE HIGH INTENSITY GROUP ON % FAT, POUNDS FAT, FAT FREE WEIGHT, WEIGHT, $\dot{V}O_2$ MAX

APPENDIX D-2. RAW DATA FOR THE LOW INTENSITY EXERCISE GROUP ON % FAT, POUNDS FAT, FAT FREE WEIGHT, WEIGHT, $\dot{V}O_2$ MAX

APPENDIX D-3. RAW DATA FOR THE CONTROL GROUP ON % FAT, POUNDS FAT, FAT FREE WEIGHT, WEIGHT, $\dot{V}O_2$ MAX

APPENDIX D-1

RAW DATA FOR THE HIGH INTENSITY EXERCISE GROUP ON % FAT, POUNDS FAT, FAT FREE WEIGHT, WEIGHT, $\dot{V}O_2$ MAX

Test Time	% Fat	Pounds Fat	Fat Free Weight	Weight (lbs)	$\dot{V}O_2$ max (ml/kg/min)
1. pre	25.90	36.45	104.05	140.50	41.80
post	24.59	34.89	107.01	141.90	38.20
2. pre	31.78	43.85	094.14	138.00	39.00
post	23.95	32.19	102.21	134.40	41.30
3. pre	27.30	36.72	097.78	134.25	38.60
post	26.60	35.67	098.13	133.80	39.00
4. pre	26.57	35.67	098.59	134.25	28.10
post	24.29	32.33	100.27	133.10	37.20
5. pre	24.54	31.35	096.40	127.75	36.30
post	23.53	29.71	096.55	126.26	38.50
6. pre	21.53	25.30	092.20	117.50	37.50
post	22.07	26.04	091.95	117.92	37.20
7. pre	34.19	42.57	081.93	124.25	31.20
post	31.78	41.41	088.88	130.24	38.90
8. pre	25.68	28.50	082.50	111.00	31.40
post	27.04	30.39	082.02	112.42	39.40
9. pre	29.05	34.06	083.19	117.25	43.10
post	24.90	29.17	087.89	117.04	41.40
10. pre	27.34	36.22	096.27	132.00	42.20
post	25.60	34.52	099.90	133.00	44.00
pre $\bar{X}$	27.39	35.07	092.05	127.70	36.92
SD	3.60	5.71	7.66	9.89	5.15
post $\bar{X}$	25.44	32.63	095.48	128.20	39.50
SD	2.66	4.27	7.63	9.47	2.13

APPENDIX D-2

RAW DATA FOR THE LOW INTENSITY EXERCISE GROUP OF % FAT, POUNDS FAT, FAT FREE WEIGHT, WEIGHT, $\dot{V}O_2$ MAX

	Test Time	% Fat	Pounds Fat	Fat Free Weight	Weight (lbs)	$\dot{V}O_2$ max (ml/kg/min)
11.	pre	20.37	22.45	087.79	110.25	32.30
	post	20.08	21.79	086.71	108.52	44.40
12.	pre	26.11	32.93	101.67	119.00	43.30
	post	27.68	30.53	107.13	116.93	42.20
13.	pre	26.74	38.91	106.59	145.50	32.00
	post	27.77	41.91	109.01	150.92	34.30
14.	pre	29.44	35.11	084.14	119.25	39.30
	post	22.16	26.72	093.88	120.60	36.30
15.	pre	26.53	33.76	093.46	127.25	38.60
	post	24.08	30.46	096.04	127.60	38.10
16.	pre	28.37	38.58	097.41	136.00	37.40
	post	26.15	35.09	099.11	134.20	35.40
17.	pre	32.61	45.74	094.51	140.25	35.20
	post	30.17	41.76	096.65	138.38	33.30
18.	pre	28.54	31.47	078.78	110.25	43.50
	post	24.08	27.28	086.02	113.30	41.00
19.	pre	25.13	30.47	090.78	121.25	30.00
	post	21.99	24.04	093.71	120.12	37.10
20.	pre	28.37	31.77	080.28	112.00	35.90
	post	28.75	32.63	080.87	113.50	43.00
	pre $\bar{X}$	27.22	34.12	091.54	124.10	36.75
	SD	3.19	6.17	9.04	12.73	4.59
	post $\bar{X}$	25.29	31.22	094.91	124.41	38.51
	SD	3.32	6.83	8.92	13.28	3.89

APPENDIX D-3

RAW DATA FOR CONTROL GROUP ON % FAT, POUNDS FAT, FAT FREE WEIGHT, WEIGHT, $\dot{V}O_2$ MAX

	Test Time	% Fat	Pounds Fat	Fat Free Weight	Weight (lbs)	$\dot{V}O_2$ max (ml/kg/min)
21.	pre	20.29	30.13	118.37	148.50	36.40
	post	20.87	30.78	116.72	147.51	32.60
22.	pre	28.11	38.93	099.57	138.50	37.20
	post	25.68	36.44	105.46	141.90	34.50
23.	pre	28.54	42.81	107.19	150.00	39.90
	post	30.61	47.89	108.57	156.46	37.30
24.	pre	21.78	24.50	087.99	112.50	44.70
	post	22.41	24.74	085.66	110.44	39.90
25.	pre	19.79	24.53	099.46	124.00	39.40
	post	22.70	28.62	097.44	126.06	32.20
26.	pre	29.31	40.48	097.55	138.00	40.30
	post	28.37	39.63	100.07	139.70	35.70
27.	pre	29.01	35.68	087.32	123.00	30.00
	post	30.09	38.06	088.44	126.50	27.30
28.	pre	37.33	59.02	108.84	173.00	35.80
	post	35.16	64.58	108.84	167.86	39.10
29.	pre	28.93	42.96	105.53	148.50	29.70
	post	30.87	45.23	101.29	146.52	29.00
30.	pre	34.94	45.33	084.44	129.75	32.20
	post	34.32	45.53	087.13	132.66	32.90
	pre $\bar{X}$	27.80	38.44	099.63	138.53	36.56
	SD	5.80	10.43	10.80	17.37	4.83
	post $\bar{X}$	28.11	40.15	099.96	139.56	34.05
	SD	5.01	11.49	10.40	16.51	4.10

APPENDIX E

APPENDIX E-1. RAW DATA OF THE HIGH INTENSITY EXERCISE GROUP ON RESTING AND POST EXERCISE LACTATE, FFA AND GLUCOSE LEVELS PRE AND POST TRAINING

APPENDIX E-2. RAW DATA OF THE LOW INTENSITY EXERCISE GROUP ON RESTING AND POST EXERCISE LACTATE, FFA AND GLUCOSE LEVELS PRE AND POST TRAINING

APPENDIX E-3. RAW DATA OF THE HIGH INTENSITY CONTROL GROUP ON RESTING AND POST EXERCISE LACTATE, FFA AND GLUCOSE LEVELS PRE AND POST TRAINING

APPENDIX E-4. RAW DATA OF THE LOW INTENSITY CONTROL GROUP ON RESTING AND POST EXERCISE LACTATE, FFA AND GLUCOSE LEVELS PRE AND POST TRAINING

APPENDIX E-1

RAW DATA OF THE HIGH INTENSITY EXERCISE GROUP ON RESTING AND POST EXERCISE LACTATE, FFA AND GLUCOSE LEVELS PRE AND POST TRAINING

	Lactate pre-pre	Lactate post-pre	Lactate pre-post	Lactate post-post	Glucose pre-pre	Glucose post-pre	Glucose pre-post	Glucose post-post	FFA pre-pre	FFA post-pre	FFA pre-post	FFA post-post
1.	28.56	32.76	49.56	52.50	60.76	68.16	63.08	57.76	0.88	0.35	0.61	1.22
2.	28.56	07.77	41.44	29.82	76.94	69.55	64.23	56.84	0.94	0.56	0.83	0.42
3.	30.24	09.24	34.44	28.56	73.24	67.92	65.85	70.47	1.31	0.46	0.84	0.48
4.	25.20	26.04	35.70	41.16	74.40	61.46	70.71	59.61	0.31	0.50	0.72	0.39
5.	21.84	21.00	26.88	27.51	67.47	55.45	55.45	73.01	0.94	0.31	1.32	0.19
6.	13.02	27.30	34.44	30.24	63.21	70.70	66.08	62.85	0.82	0.30	0.78	0.39
7.	10.50	15.54	45.36	35.28	72.78	65.39	70.70	61.92	0.66	0.43	0.38	0.70
8.	13.86	57.96	21.84	63.00	71.97	65.62	64.23	41.13	0.48	0.44	0.52	0.39
9.	10.92	10.92	39.20	34.86	70.71	60.54	67.24	75.32	0.40	0.42	0.81	0.24
10.	20.16	06.30	48.72	23.94	71.86	73.01	61.23	67.70	0.12	0.58	0.66	0.58
$\bar{X}$	20.27	21.48	37.76	36.69	70.33	65.78	64.88	62.66	.686	.435	.747	.500
SD	7.75	15.73	9.01	12.33	5.06	5.31	4.50	9.22	.359	.009	.249	.293

APPENDIX E-2

RAW DATA OF LOW INTENSITY EXERCISE GROUP ON RESTING AND POST EXERCISE LACTATE, FFA, AND GLUCOSE LEVELS PRE AND POST TRAINING

	Lactate pre-pre	Lactate post-pre	Lactate pre-post	Lactate post-post	Glucose pre-pre	Glucose post-pre	Glucose pre-post	Glucose post-post	FFA pre-pre	FFA post-pre	FFA pre-post	FFA post-post
11.	27.30	23.94	32.76	21.42	80.87	75.79	73.48	58.69	0.31	0.41	0.72	0.35
12.	12.60	25.20	13.02	39.90	90.81	61.69	78.10	62.15	0.85	0.45	0.70	0.30
13.	22.68	25.41	26.88	26.25	73.01	65.62	71.17	72.09	0.40	0.33	1.33	1.15
14.	18.48	33.60	33.60	36.12	71.16	74.17	65.16	68.39	0.76	0.37	1.04	0.34
15.	12.88	24.36	15.12	29.40	79.95	68.86	70.01	68.86	0.78	0.45	0.75	0.34
16.	30.24	20.16	62.16	38.22	73.01	61.00	63.77	58.68	0.81	0.43	0.86	0.81
17.	18.88	32.34	31.92	39.06	73.02	72.78	71.63	72.78	0.60	0.73	0.64	0.32
18.	16.80	21.00	35.28	24.36	74.98	68.16	69.60	66.78	0.86	0.37	1.18	0.77
19.	21.00	23.73	40.32	33.60	73.94	70.00	72.09	66.50	0.70	0.16	1.13	0.32
20.	18.68	32.77	33.60	26.46	69.55	75.78	66.54	69.32	0.66	0.59	0.66	0.50
$\bar{X}$	19.91	26.25	32.47	31.48	76.03	69.39	70.16	66.42	.673	.429	.901	.520
SD	5.66	4.89	13.60	6.74	6.29	5.40	4.23	5.04	.188	.150	.249	.292

APPENDIX E-3

RAW DATA OF THE HIGH INTENSITY CONTROL GROUP ON RESTING AND POST EXERCISE LACTATE, FFA AND GLUCOSE LEVELS PRE AND POST TRAINING

	Lactate pre-pre	Lactate post-pre	Lactate pre-post	Lactate post-post	Glucose pre-pre	Glucose post-pre	Glucose pre-post	Glucose post-post	FFA pre-pre	FFA post-pre	FFA pre-post	FFA post-post
21.	19.32	24.78	40.52	42.84	68.85	76.48	72.55	78.33	0.30	0.70	0.28	0.59
22.	31.90	34.44	36.96	89.04	75.33	71.86	77.17	76.48	0.72	0.75	0.65	0.79
23.	21.00	37.30	43.68	34.89	70.93	67.93	69.55	59.61	0.86	0.52	0.52	0.31
24.	31.90	32.84	52.08	52.92	77.86	69.77	75.85	67.24	0.12	0.28	0.52	0.54
25.	15.12	19.98	36.75	59.22	59.23	60.53	66.31	64.06	0.86	0.58	1.00	0.95
26.	36.96	37.80	60.06	55.44	61.69	65.39	52.52	66.54	0.84	0.50	0.92	0.30
27.	17.22	20.50	43.56	60.90	82.26	71.63	79.71	67.01	0.61	0.64	1.16	0.76
28.	12.54	11.60	41.44	49.14	62.23	61.23	78.09	72.99	0.57	0.59	0.41	0.77
29.	26.88	33.60	45.36	39.48	79.25	62.23	72.09	64.00	0.44	0.30	0.65	0.63
30.	20.16	19.32	53.76	73.50	68.40	73.50	70.01	61.92	0.64	0.34	0.54	0.45
ΣX	233	272.16	454.71	557.37	703.03	680.55	713.85	678.12	5.96	5.20	6.63	6.09
$\bar{X}$	23.3	27.22	45.47	55.74	70.30	68.06	71.39	67.81	.596	.52	.663	.609
SD	8.15	9.12	7.73	16.26	8.49	5.53	7.86	6.20	.248	.166	.278	.214

APPENDIX E-4

RAW DATA OF THE LOW INTENSITY CONTROL GROUP ON RESTING AND POST EXERCISE LACTATE, FFA AND GLUCOSE LEVELS PRE AND POST TRAINING

	Lactate pre-pre	Lactate post-pre	Lactate pre-post	Lactate post-post	Glucose pre-pre	Glucose post-pre	Glucose pre-post	Glucose post-post	FFA pre-pre	FFA post-pre	FFA pre-post	FFA post-post
31.	12.60	33.18	32.50	39.90	76.25	70.48	85.02	73.48	0.42	0.46	0.48	0.72
32.	21.84	32.00	48.72	60.06	78.33	71.40	55.02	50.06	0.34	0.86	0.42	1.00
33.	21.70	15.54	26.60	32.76	75.85	67.03	77.86	61.03	0.60	0.58	0.30	0.54
34.	31.05	37.80	32.55	36.96	77.40	64.37	76.48	64.00	0.22	0.94	0.11	0.50
35.	28.56	24.10	42.84	37.38	63.12	61.46	63.77	69.78	0.44	0.25	0.79	0.54
36.	19.32	36.96	28.56	49.00	62.40	62.85	62.85	56.38	0.65	0.75	0.88	0.68
37.	21.00	23.94	36.96	31.08	82.50	72.09	70.80	70.70	0.58	0.38	0.81	0.37
38.	28.56	15.96	35.28	20.00	74.63	66.31	70.24	72.78	0.68	0.16	1.30	0.21
39.	12.60	12.80	17.92	16.90	78.33	64.47	72.55	64.58	0.89	0.27	0.52	0.52
40.	20.16	19.50	42.00	50.40	68.40	73.47	63.77	62.85	0.48	0.78	0.76	0.63
ΣX	217.39	251.78	343.93	374.44	737.21	673.93	698.36	645.64	5.3	5.43	6.37	5.71
$\bar{X}$	21.74	25.18	34.39	37.44	73.72	67.39	69.84	64.56	.53	.543	0.637	.571
SD	6.29	9.28	8.91	13.37	6.78	4.21	8.75	7.49	.191	.279	.341	.211

APPENDIX F

TRAINING HEART RATES OF THE LOW INTENSITY EXERCISE GROUP AT 65% OF MAXIMUM HEART RATE AND THE HIGH INTENSITY EXERCISE GROUP AT 80% OF MAXIMUM HEART RATE

APPENDIX F

TRAINING HEART RATES OF THE LOW INTENSITY EXERCISE GROUP AT 65% OF MAXIMUM HEART RATE AND THE HIGH INTENSITY EXERCISE GROUP AT 80% OF MAXIMUM HEART RATE

	Low Intensity Exercise Group	High Intensity Exercise Group
	125	162
	116	155
	129	151
	129	163
	119	146
	126	152
	133	150
	129	158
	125	158
	125	156
$\bar{X}$	125.60	155.10
SD	5.02	5.40

APPENDIX G

EXAMPLES OF CHANGES IN THE AVERAGE Kp LOAD TO ELICIT A RELATIVE INTENSITY HEART RATE OF 65 OR 80% MAXIMUM HEART RATE FOR EACH WEEK OF TRAINING

APPENDIX G

EXAMPLES OF CHANGES IN THE AVERAGE Kp LOAD TO ELICIT A RELATIVE INTENSITY HEART RATE OF 65 OR 80% MAXIMUM HEART RATE FOR EACH WEEK OF TRAINING

Week	Subject A (65%)	Subject B (80%)
1	.89	1.75
2	.79	1.72
3	.99	1.86
4	.98	1.95
5	1.04	1.92
6	1.00	1.99
7	.99	1.99
8	1.03	1.84
9	1.15	2.12

APPENDIX H

EXAMPLES OF CHANGES IN THE AVERAGE TRAINING TIME IN MINUTES PER WEEK FOR HIGH AND LOW INTENSITY EXERCISE GROUPS AT RELATIVE INTENSITIES OF 65 OR 80% OF MAXIMUM HEART RATE

APPENDIX H

EXAMPLES OF CHANGES IN THE AVERAGE TRAINING TIME IN MINUTES PER WEEK FOR HIGH AND LOW INTENSITY EXERCISE GROUPS AT RELATIVE INTENSITIES OF 65 OR 80% OF MAXIMUM HEART RATE

Week	Subject A (65%)	Subject B (80%)
1	36.67	19.83
2	40.83	24.33
3	31.83	18.00
4	35.33	18.00
5	36.33	19.50
6	40.67	19.83
7	38.67	19.83
8	37.00	19.17
9	35.00	19.00

APPENDIX I

INFORMED CONSENT FORM

Consent For Exercise Tests

I, ____, hereby agree to undertake an exercise stress test, administered twice, designed to determine my maximal oxygen consumption. The test will be conducted in the presence of a cardiac nurse. Heart rate will be monitored throughout the test. I understand that I will perform a relative workload test during which blood samples will be drawn before and after exercise by a qualified technician. Heart rate will be monitored throughout. I understand that body composition will be determined by hydrostatic weighing.

Every effort will be made to conduct the tests in such a manner as to minimize discomfort and risk. However, I understand that with any type of exercise test there are potential risks, and at any time during the test that I experience unusual discomfort, I will ask to discontinue the test.

In agreeing to such an examination, I waive any legal recourse against the University of Alberta from any and all claims resulting from this fitness test.

DATE ________________________ SUBJECT ________________________
Signature

WITNESS ______________________